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CHLORINATED BENZENES IN THE AQUATIC ENVIRONMENT

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Prepared for

WATER RESOURCES BRANCH

Ontario Ministry of the Environment

by

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SUMMARY

In formulating the following Provincial Water Quality Objectives for chlorinated benzenes their production and uses, chemical and physical characteristics, interactions and persistence in the aquatic environment, and toxicity to aquatic biota have been considered.

Quantitative Structure-Activity Relationships (QSAR), based on aquatic toxicity data and hydrophobicity (as measured by octanol-water partition coefficients, log K_{ow}), were used to produce estimated toxicity data where experimental data were scanty or absent. As well, QSAR methodology was used to interpret and generally rationalize the development of objectives. This allowed objectives to be developed for all chlorinated benzene compounds and their isomers.

Although objectives are usually considered to be primarily for the protection of aquatic life, it is not reasonable to propose objectives that might impair other uses. Since there is sufficient information on bioaccumulation hazard to suggest that other uses might be affected at levels judged acceptable in terms of toxicity to aquatic biota alone, consideration of bioaccumulation hazard has been incorporated in the objectives development process.

A summary of the proposed Provincial Water Quality Objectives for chlorinated benzenes follows.

		Objective (µg/L)
Monochlorobenzene		15
Dichlorobenzene	1,2	2.5
	1,3	2.5
	1,4	4.0
Trichlorobenzene	1,2,3	0.90
	1,2,4	0.50
	1,3,5	0.65
Tetrachlorobenzene	1,2,3,4	0.10
	1,2,3,5	0.10
	1,2,4,5	0.15
Pentachlorobenzene		0.030
Hexachlorobenzene		0.0065

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section 1

SOURCES, OCCURRENCE, AND CHARACTERISTICS

1.0 SOURCES, OCCURRENCE, AND CHARACTERISTICS

1.1 Production and Uses

1.1.1 Production

Chlorinated benzenes are manufactured chemicals which are synthesized directly or produced as a byproduct of various chemical processes. There are no known natural sources of chlorinated benzenes.

A brief outline concerning the production and uses of chlorinated benzenes has been prepared and included in this document; specifically, information on how these chemicals are produced, how much is produced, and where. As well, information on how they are used, how much is allocated to the various types of uses and where such uses might occur, is of significance and is also included.

A brief historical perspective is included to allow the time course and extent of the presence of chlorinated benzene in the environment to be evaluated. Finally, indications of future trends in production and use, where available, are presented to assist in delineating areas of increasing and/or decreasing environmental concern.

Chlorinated benzenes were initially manufactured, for industrial purposes, in England in 1909. In the United States, production began in 1915 and was greatly increased during World War 1 when the demand for phenol grew; chlorinated benzenes being intermediates in the production of phenols (MOE, 1978). Sediment core data from Lake Ontario indicates

that most of the chlorinated benzenes entered the lake after 1941 (Oliver and Nicol, 1982). Presently, the United States, West Germany, the United Kingdom, France and Japan are the principal chlorobenzene-producing countries (Hardie, 1974). Current production estimates, where available, are presented in Table 1.

Monochlorobenzene (MCB) is produced by direct chlorination of benzene at an elevated temperature in the presence of a catalyst, usually ferric chloride (MOE, 1978). Dichlorobenzenes (DCB) are produced as a byproduct of MCB production. The crude dichlorobenzenes are isolated from the MCB by distillation and separated into isomers by fractional distillation or crystallization. Hexachlorobenzene (HCB) is rarely directly produced, as it is a byproduct of numerous chemical processes. Other chlorobenzenes are obtained by further chlorination of lesser chlorinated ones or reduction of higher chlorobenzenes. The production of specific chlorobenzenes can be maximized by manipulation through use of catalysts or by varying reaction conditions. However, it is not possible to avoid co-production of higher isomers during reactions (Ware and West, 1977).

MCB was the first organic chemical produced in large quantities and is still of the greatest commercial importance (Ware and West, 1977). MCB accounts for 600,000 tonnes of the estimated worldwide production of 900,000 tonnes of halobenzenes annually (WHO, 1980). Production of MCB has decreased in the United States as industry has shifted towards using cumene as an intermediate for phenol and as the use of DDT has been restricted. There is no manufacturer of monochlorobenzenes in Canada. At present, Record Chemical

Co., Inc. with plants in Quebec and British Columbia is apparently the only importer (MOE, 1978).

The commercially important dichlorobenzenes - 1,2 DCB and 1,4 DCB - are almost entirely produced by distilling DCB contaminants from MCB. In the United States, nearly 100 million pounds (45,300 tonnes) of these isomers are produced annually. In Canada, the Record Chemical Inc. imports crude DCB from which 1,2 and 1,4-DCB are distilled (MOE, 1978).

The yearly production of 1,2,4-trichlorobenzene (TCB) and 1,2,4,5-tetrachlorobenzene (TeCB) is 12,700 and 8,165 tonnes annually in the United States (Ware and West, 1975; Lewis, 1975). HCB is produced in sizeable quantities as a byproduct of chemical processes. Of the estimated 3,900 tonnes produced in this manner, about 318 tonnes are purified for commercial uses (Quinlivan et al., 1975; Mumma and Lawless, 1975). The remaining chlorobenzenes have limited commercial value and are neither produced directly nor in large quantities.

As MCB is being phased out as an intermediate for phenol, the production of MCB in the United States has decreased and is expected to continue to drop. The future market for 1,2-DCB in the United States is expected to expand while the 1,4-DCB market is considered saturated (Ware and West, 1977).

Several uses of chlorinated benzenes as chemical intermediates are in the decline, not because of environmental concern surrounding chlorobenzenes, but due to the toxicity of the final product or its impurities (Ware and West, 1977). Chlorobenzenes may be replaced in dielectric fluids, heat

transfer fluids or as solvents in the future if more economical, less toxic or less corrosive alternatives are found. However, the chlorobenzenes may have new applications as they were discovered to Q switch the carbon dioxide laser. Also, pentachlorobenzene (QCB) and HCB could be used in the future as precursors in the production of tractable polyamideimide and polyimides, and thermosetting polyimides (Kwiatkowski et al., 1976).

Imports of chlorinated benzenes have been rising steadily in Canada recently, and it is expected that this trend will continue, particularly for 1,4-DCB. There is an oversupply of chlorobenzene plants in North America and therefore it is expected that Canada will continue as an important rather than becoming a producer of chlorobenzenes (MOE, 1978).

A final observation on the production of chlorobenzenes comes from Glase and Henderson (1975) and Mori et al. (1978). They reported that chlorination of municipal sewage effluent containing small amounts of benzene and benzene derivations, produced low levels of a variety of chlorinated benzenes. The overall significance of inadvertent production by this means to general environmental contamination is not known but it may be a source of at least part of the low concentrations of chlorinated benzenes reported as present in a number of sewage treatment plants in Ontario (Munro et al., 1983).

1.1.2 Uses

The sources of chlorobenzene contamination are numerous as its uses are varied and frequently dissipative. Chlorinated benzenes can enter the environment as solvents, pesticides,

dielectric fluids, deodorants, and chemical contaminants or intermediates (see Table 1). Chlorinated benzenes are prevalent both in solid and liquid industrial waste and atmospheric discharges. The differentiation and assessment of sources is compounded by the large worldwide production of particular chlorobenzene compounds and isomers.

Initial loss of monochlorobenzene (MCB) occurs during its production (Ware and West, 1977). Further losses occur as MCB evaporates from final products and industrial wastes where it is a contaminant (Ware and West, 1977). An estimated 300,000 tonnes of MCB are released into surface waters and the atmosphere worldwide annually (WHO, 1980). A significant quantity of MCB is used as a solvent and an insecticide. In Canada small quantities are imported for use as a speciality solvent and heat exchange medium (MOE, 1978). Other potential sources of MCB contamination in Canada include atmospheric losses from various chemicals and processes where MCB is a contaminant and from imported products containing MCB as a solvent base.

The dichlorobenzenes (DCB) are present as contaminants of MCB and therefore may accompany MCB contamination, varying with the purity of MCB. 1,2-DCB is both volatile and heavily used as a solvent giving rise to an annual loss of approximately 40,000 tonnes worldwide (WHO, 1980). In Canada, small quantities are imported for use in metal cleaning, rustproofing and for minor uses as a solvent in lacquers, resins and insect repellant (MOE, 1978). The primary use of 1,4-DCB, as a household and industrial deodorant, makes it a common contaminant. An estimated 80,000 tonnes of 1,4-DCB enter surface water and the atmosphere annually worldwide (WHO,

1980). In Canada, it is imported for use as a household deodorizer and moth repellant (MOE, 1978). While 1,3-DCB may occur as a contaminant of other DCB's, it has little commercial use.

Trichlorobenzene (TCB) and tetrachlorobenzene (TeCB) are used primarily as intermediates in chemical processes but they also occur in unknown quantities as impurities in DCB (WHO, 1980). 1,2,4-TCB serves as a dye carrier for polyester material, as dielectric fluid and as an intermediate for various chemical processes. 1,2,4,5-TeCB is used primarily as a chemical intermediate. These isomers may escape into the environment in chemical effluent or as leakage from defective transformers and capacitors.

Pentachlorobenzene (QCB) has very limited commercial use in North America; in Europe it is used in limited quantities to produce the pesticide pentachlorobenzene. It is coproduced with hexachlorobenzene and occurs at a rate of 0.02 to 3.1 per cent of the HCB produced (Villaneueva et al., 1974).

Most Hexachlorobenzene (HCB) is generated as a byproduct of chemical processes such as production of perchlorethylene, carbon tetrachloride and trichloroethylene. Detailed information on HCB in Canada appears in Tattle (1979) and DOE/NHW (1979). Losses to the environment from HCB-containing industrial waste are high (Mumma and Lawless, 1975). HCB is also present as a contaminant in the final products.

In 1945, HCB was introduced into the United States as a fungicide for cereal and seed treatment. Annually, 700,000 (318 tonnes) pounds are directed towards pest control in the

United States (National Academy of Science, 1974). HCB is presently not a registered fungicide in Canada; however, it is considered the only acceptable chemical control for dwarf bunt of wheat and is eligible for speedy registration should the need arise (Taylor, personal communication).

Evidently, certain chlorobenzenes pose greater threats of contamination to the environment, due to the dispersive nature of their uses or their presence in industrial effluent. Even when used as intermediates in a closed system, some chlorinated benzenes are lost as product contaminants or in the industrial effluent. Approximately one half of the world MCB production enters the surface waters and the atmosphere yearly (WHO, 1980). Fifty per cent of the 1,2-DCB produced in America is used in a dissipative manner. Ninety per cent of the 1,4-DCB used annually in the United States and nearly all in Canada are consumed in non-intermediate, dispersive uses (Ware and West, 1977; MOE, 1978). Most of the HCB produced as an unwanted byproduct escapes in industrial wastes.

Clearly, MCB, 1,2-DCB, 1,4-DCB, and HCB are likely to be the most widespread in the environment. Of the remaining chlorinated benzenes, 1,2,4-TCB and 1,2,4,5-TeCB, because of their numerous commercial applications, are most likely to escape into the environment.

1.2 Chemical and Physical Characteristics

Chlorinated benzenes are compounds in which one or more hydrogens on a benzene ring are replaced by chlorine. The chlorination of the benzene ring yields 12 different compounds

representing all possible combinations of direct substitution by chlorine. These are: monochlorobenzene (C_6H_5Cl), three isomers of dichlorobenzene ($C_6H_4Cl_2$), three isomers of trichlorobenzene ($C_6H_3Cl_3$), three isomers of tetrachlorobenzene ($C_6H_2Cl_4$), pentachlorobenzene (C_6HCl_5), and hexachlorobenzene (C_6Cl_6).

At 20°C, MCB, 1,2-DCB, 1,3-DCB and 1,2,4-TCB are colourless liquids. The remaining compounds are white crystalline solids at this reference temperature. All compounds have pleasant aromatic odours with the exception of 1,4-DCB which exhibits a penetrating camphoric odour.

The chemical and physical characteristics of chlorinated benzenes are summarized in Table 2. As the degree of chlorination increases, there is a corresponding increase in molecular weight and density.

Many of the properties of chlorinated benzenes tend to be related to the degree of chlorination and hence molecular weight of the compounds. Boiling points and melting points increase with increasing molecular weight. Vapour pressures and air-water partition coefficients (Henry's Law Constants) decrease as the molecular weight increases, such that the lower the degree of chlorine substitution, the greater the volatility of the pure compounds (Section 1.4.2).

Water solubilities of the chlorinated benzenes decrease progressively as the molecular weights increase. Table 2 shows both measured and calculated water solubilities. The first set of measured and reported ranges of solubilities was retrieved from the literature as a part of this study. The

second set of measured solubilities is the mean of values reported in the comprehensive review of Mackay and Shiu (1981). The corrected solubilities (in moles per cubic metre) are the actual solubility of the material if it is a liquid or the estimated subcooled liquid solubility derived from the actual solid solubility if it is a solid.

The correction is based on the method of Mackay (1982) who notes that solids dissolved in liquids tend to exhibit characteristics better described by treating it as a subcooled liquid rather than a solid. Table 2 shows there is general agreement between measured and calculated solubilities and demonstrates that the water solubilities of the chlorinated benzenes decrease progressively as the molecular weight and degree of chlorination increases.

The chlorinated benzenes are generally good solvents for fats, waxes, oils and greases (U.S. EPA, 1980a). Their lipophilic nature is indicated by their respective log K_{ow} values. Log K_{ow} is the logarithm of the octanol/water partition coefficient and is a commonly used measure of a compound's hydrophobicity or tendency to partition to a non-polar phase of a polar/non-polar mixture. In Table 2, the first set of measured values was judged by Oliver and Niimi (1983) to represent the best of the measured data available. The first set of calculated values was calculated using the "fragment method" of Leo et al. (1971) by Neely et al. (1974) and Smith (1981).

The second set of calculated log K_{ow} values was calculated by the method of Mackay (1982); based on the mean of the measured values reported by Mackay and Shiu (1981) in

Table 2. Table 2 shows that both observed and calculated log K_{ow} values increase with the higher molecular weight compounds. The higher molecular compounds would therefore be expected to have a greater potential to accumulate in the aquatic ecosystem (Section 2.2).

The chlorinated benzenes tend to be very chemically stable in terms of most ionic mechanisms; although, as will be discussed later, they are considered to be moderately reactive by free-radial pathways (Callahan et al., 1979). The resistance of chlorinated benzenes to degradation increases with increasing molecular weight; HCB being the most chemically stable of these compounds (Section 1.4.1).

As the discussion above implies, the chlorinated benzenes may be viewed as a family of similar compounds with differences in physical and chemical characteristics such as volatility, solubility, partitioning potential and stability directly related to the degree of chlorination or molecular weight of the compounds.

1.3 Chlorinated Benzenes in the Environment

Chlorinated benzenes are essentially ubiquitous in the environment as a result of their varied and frequently dissipative uses, as discussed in Section 1.1. Tables 3, 4, 5 and 6, which deal with water, sediment, aquatic biota, and birds, respectively, report on the range of concentrations encountered in the environment. Data for the Niagara River, an important Ontario river that flows through a highly industralized area known to have organic chemical pollution problems, is summarized in a separate table, Table 7. As can

be seen upon inspection of these tables, the information is primarily from North American sources, although limited amounts of European data are also included.

It is immediately obvious that there is a dearth of data on MCB. Given the fact that more of this compound is produced and used than any of the other chlorinated benzenes (see Section 1.1), the absence of data appears odd. The reason for this is twofold. Firstly, due to the high volatility and low K_{ow} (see Table 2) of this compound, it evaporates more rapidly than the other chlorinated benzenes. Therefore, it probably has the shortest half-life in water (see Table 8) and is considered to be the least likely of the group to be an aquatic problem (see Section 1.4). Secondly, due to the volatile nature of MCB, the analytical technique employed in its quantification is different than that used for the other chlorinated benzenes. It is classified as a purgable aromatic and requires equipment of a more sensitive nature for accurate detection and quantification (U.S. EPA, 1979b).

Although information suggests that point sources such as solid and liquid industrial wastes (Mumma and Lawless, 1975; Ware and West, 1977), as well as sewage treatment plants (see Table 3, also Munro et al, 1983) and other water-mediated transport, are important in environmental dispersal, long range airborne transport of chlorinated benzenes cannot be ignored. Although the current information is primarily data for HCB, atmospheric transport of the other chlorinated benzenes is also likely, although not necessarily to the same extent. HCB has been detected in rain and snow collected in the Great Lakes basin (Strachan and Huneault, 1979). This method of transport explains the presence of HCB in lake

trout from Siskiwit Lake on Isle Royale, an island in Lake Superior far removed from direct human influence (Swain, 1978). In fact, Eisenreich et al. (1981) have estimated that deposition from airborne HCB is about 1.7, 1.2, 1.2, 0.53, and 0.39 tonnes per year for Lakes Superior, Michigan, Huron, Erie and Ontario, respectively.

As discussed in Section 1.1.2, MCB, 1,2-DCB, 1,4-DCB and HCB are likely to be the most prevalent followed by 1,2,4-TCB and 1,2,4,5-TeCB. The remainder of the group would be expected to be less abundant. This trend is reflected in a general way in the water data (Table 3) although some of the compounds in the less abundant group are detected in many areas. Therefore, their production (as by-products) and release to the environment cannot be dismissed as insignificant.

Although there is a general trend to lower reported water concentrations for more highly chlorinated compounds, in the organic component of the aquatic environment, as a rule, the opposite is true. The sediment, bird, and, especially the aquatic biota data indicate that the more highly chlorinated compounds are present at higher levels (see Sections 1.2, 1.4 and 2.2), and might, therefore, be expected to be more problematic.

For Ontario, data on geographical occurrences are limited, thereby precluding the production of a comprehensive list of potential problem areas. However, based on the location of chemical manufacturing industries in the St. Clair River and the Niagara River areas, these areas, as well as the downstream lakes, Lake Erie and Lake Ontario (especially the latter), would be potential areas of concern.

1.4 Interaction and Persistance in the Environment

1.4.1 Degradation

Chlorinated benzenes are relatively resistant to chemical, biological and photolytic degradation and hence tend to be persistent in aquatic ecosystems. The resistance to degradation increases with the more highly chlorinated isomers.

Abiotic Degradation

Very little specific information pertaining to photolysis of chlorinated benzenes in the aqueous or atmospheric environments under ambient conditions exists. However, the hydroxy radicals (HO) that produce much of this type of reaction are readily produced by photochemical processes in the atmosphere in sufficient quantities to react with chlorobenzenes in the air (MacGregor, personal communication).

Dilling et al. (1976) studied the photodecomposition rates of a number of chlorinated hydrocarbons in a simulated atmosphere. MCB was found to decompose relatively slowly compared to other chlorinated compounds, however, in the presence of nitric oxide, a probable reaction initiator, 43% of the initial 10 ppm concentration underwent reaction in 7.5 hours. This translates to a half-life of 8.7 hours, however, this probably is not representative of the photooxidation half-life of MCB under environmental concentrations and conditions.

Brown et al. (1975) report that DCB are reactive to HO radicals in air, with half-live of approximately 3 days. HCB is

moderately reactive to HO in air with a half-life of approximately 2 days, pentachlorophenol being the reaction product. Simmons et al. (1976), report 1,2,4,-TCB is also susceptible to attack by HO radicals in air with an estimated half-life in the order of one to several days.

Plimmer and Klingebiel (1976) investigated the photodecomposition of crystalline HCB and a hexane solution of HCB when exposed to 4.5 hour exposures to sunlight, to a 40 watt sunlamp and to fluorescent lighting. The experiments indicated that photolysis of HCB was extremely slow. In the presence of diphenylamine, the photodecomposition of HCB was sensitized. They concluded that naturally occurring organic complexes (e.g. humic acids) in the aqueous environment may serve as photosensitizers and enhance photolysis. However, no studies have been carried out on photodegradation of HCB in the presence of these possible photosensitizers.

Solutions of HCB in both hexane and benzene (1 µg/10 ml) were irradiated by Laseter et al. (1976) at varying time intervals of 30, 65 and 120 minutes. Irradiations were conducted at 253.7 mm. After 60 minutes of irradiation in benzene less than 10% of the original HCB remained. However, based on the fact that the aforementioned wavelength is below those naturally occurring in the atmosphere, photodecomposition is probably not an important fate process for HCB in the aquatic environment (Callahan et al., 1979).

There is limited information pertaining to oxidation of chlorinated benzenes in the aquatic environment. Most of the available data applies to the atmospheric environment.

Brown et al. (1975), report both 1,2-DCB and 1,4-DCB are resistant to autooxidation by the peroxy radical (RO_2) in water and by ozone (O_3) in air. HCB is also unreactive towards O_3 and RO_2 in air. Callahan et al. (1979) report that oxidation of HCB would not be expected to be an important fate under ambient conditions since the stability of HCB is such that oxidation of HCB would occur only under the most extreme conditions.

There is limited data available on the hydrolysis of chlorinated benzenes in the aquatic environment. Chlorinated benzenes being aryl halides typically are characterized by very low reactivity to nucleophilic reagents such as OH and, therefore, undergo nucleophilic substitution only with extreme difficulity i.e. strong bases and high temperatures required (Morrison and Boyd, 1971). Consequently hydrolysis of chlorinated benzenes under environmental conditions would not be expected to be a major consideration (Callahan et al. 1979).

Morrison and Boyd (1971) report MCB is converted to phenol by aqueous sodium hydroxide only at temperatures in excess of 300°C. Patai (1973) reports temperatures of at least 250°C are required for the nucleophilic substitution of HCB to its pentachlorophenyl derivatives in the presence of aqueous ammonia. Similarly Leoni and D'Arca (1976) concluded HCB is chemically very inert at room temperature and temperatures in the range of 130-200°C are required for reactions with caustic alkalis to proceed.

Biotic Degradation

In general, chlorinated benzenes in the environment may be biologically degraded; however, the extent to which they are broken down varies greatly, with the higher chlorinated isomers exhibiting greater resistance to biodegradation (Ware and West, 1977). This section considers only microbial biodegradation. Biotransformation and elimination in aquatic biota are discussed in greater detail in Section 2.2.

Gibson et al. (1968) reported that MCB could be oxidized by the microorganism Pseudomonas putrida only when they are already growing on an aromatic carbon source; in these experiments toluene was the initial growth substrate.

Using the BOD (Biochemical Oxygen Demand) test as an indication of the biodegradability of pure compounds, Heukelekian and Rand (1955) showed a relationship between BOD and the structure of a chemical. The presence of a chlorine atom on a benzene ring appears to lower the BOD value indicating an increased resistance to microbial attack. The 5 day BOD of MCB was reported as 0.03 g/g while the 10 day BOD of benzene was reported as 1.20 g/g. Although the difference in the incubation times may account for some of the reported difference in the BOD values, Alexander and Lustigman (1966) also found the rate of biodegradation of the benzene ring to be retarded by the presence of a chlorine atom.

Based on the findings reported above and the greater resistance of highly halogenated compounds to biodegradation (Ware and West, 1977), Callahan et al. (1979) presumed the other chlorinated benzenes, being more chlorinated than MCB, would

biodegrade less rapidly than MCB under the same conditions of exposure to microorganisms.

Thom and Agg (1975) presented evidence that 1,2-DCB, 1,3-DCB, 1,4-DCB are persistant synthetic organic chemicals which are unlikely to be removed during biological sewage treatment even after prolonged exposure to the microorganisms. However, MCB is listed by Thom and Agg (1975) as a chemical which should undergo degradation in biological sewage treatment if the organisms are provided a suitable acclimitization period.

Other reports, document biodegradation of DCB. Midwest Research Institute (1974) reported that 1,4-DCB is degraded to 2,5-dichlorophenol, dichloroquinol and is conjugated by biological organisms. Gubser (1969) reported sewage sludge organisms degraded 1,2-DCB. No degradation products were given.

Biodegradation of 1,2,4-TCB was investigated by Simmons et al. (1976) using unacclimatized industrial wastewater microorganisms. About 99% of the initial 1.7 mg/L concentration of 1,2,4-TCB disappeared after 10 days. However, the BOD value indicated only 55% of the theoretical BOD value was removed. The remaining theoretical demand was attributed to incompletely oxidized metabolites of 1,2,4-TCB which were probably incorporated in the cell wall. Using a higher initial concentration of 2.6 mg/L, the rate of 1,2,4-TCB biodegradation was slower for the first few days (28% removal after 7 days versus 43% removal at the lower concentration) however, after 10 days complete removal still occurred.

In another phase of these experiments Simmons et al. (1976) studied the formation rate of $^{14}CO_2$ (Carbon dioxide) through biodegradation of 1,2,4-TCB by activated sludge. After 5 days, 13% of the TCB remained with 56% conversion to carbon dioxide and 23% conversion to polar metabolites. Seven percent of the TCB evaporated. Approximately 75% of the TCB remaining in the water was associated with the solids in the waste sludge. This accounted for the low volatility from the system.

Haas et al. (1974) reported only a slight decrease in concentration (cells and water) of 1,2,4-TCB (initial concentration 50 mg/L) after 50 hours of exposure to pre-conditioned treatment plant cultures. Approximately 40% of the TCB was absorbed by the organisms; however, this absorbed fraction was not substantially biodegraded.

Beck and Hansen (1974) found the half-lives QCB and HCB in soil (incorporated at 10 kg/ha) stored in plastic-covered pots averaged 270 days and 4.2 years, respectively. Isensee et al. (1976) studied soil treated with 0.1 to 100 ppm of HCB stored in covered containers for 12 months under aerobic (sterile and nonsterile) and nonsterile anaerobic conditions. Under the treatment rates and storage conditions tested no soil incorporated HCB was lost indicating HCB is very persistent in soil if evaporation is controlled. Beall (1975) reported in a simulated pasture experiment a 55% HCB loss from the 2 cm surface soil in 2 weeks, with no HCB loss from the 2 to 4 cm depth during 19 months.

Based on the above research, evaporation of HCB and not biodegradation appears to be a major loss mechanism of HCB

from soil. Volatility of chlorinated benzenes and its role in the environmental fate of these compounds is discussed in more detail in the following section.

1.4.2 Evaporation

Data on the evaporation of chlorinated benzenes are limited. The data that are available represent a diverse range of conditions, some of which are not directly representative of those found in the aquatic environment. Air-water partition coefficients (Henry's Law Constants), calculated from laboratory data by Mackay and Shiu (1981) and vapour pressures are reported in Table 2.

However parameters such as partitioning to sediment, degree of mixing, and water column depth are important considerations governing the in situ evaporation half-lives of these compounds and their ultimate fate in the aquatic environment.

Garrison and Hill (1972) reported that MCB, 1,2-DCB, 1,4-DCB and 1,2,4-TCB at concentrations of 300, 100, 300 and 100 mg/L, respectively, evaporated almost completely (less than 1 mg/L remained) from aerated distilled water in less than 4 hours. In unaerated distilled water using the same initial concentrations, MCB, 1,2-DCB and 1,4-DCB volatilized to less than 1 mg/L remaining in less than 3 days. The 1,2,4-TCB disappeared in less than 2 days.

Using the data of Garrison and Hill (1972), Callahan et al. (1979) estimated the evaporation half-lives of the isomers, assuming first order kinetics were applicable and that gas

stripping and adsorption onto the container walls were not contributing to the observed removals.

Callahan et al. (1979) calculated the following half-lives in water:

	Evaporation Half-Life	
Isomer	Aerated	Quiescent
MCB	30 minutes	9 hours
1,2-DCB	<40 minutes	<9 hours
1,3-DCB	<30 minutes	<9 hours
1,2,4-TCB	36 minutes	7.2 hours

Schwarzenbach et al. (1979) conducted a one year study of 1,4-DCB in Lake Zurich, Switzerland. Sewage effluents were the major input sources of the isomer. An average annual mass transfer coefficient of 1 cm/hr was estimated for the transport of 1,4-DCB from the lake to the atmosphere. Mass transfer to the atmosphere was determined as the predominant elimination process. Of the 60 kg of 1,4-DCB eliminated from the lake in the course of the year, only 2 kg was attributed to elimination by partitioning to particulate matter and subsequent sedimentation.

Ware and West (1977) cite the work of Simmons et al. (1976) who calculated the evaporation rate constant of 1,2,4-TCB from water to be 9.3 cc/hr. No further experimental information was provided; however, Ware and West (1977) estimated this rate to correspond to a half-life of 45 minutes in water 10 cm deep measured at standard temperature and pressure.

Schauerte et al. (1982) maintained the concentration of HCB at 50 µg/L in a small experimental pond. Five dosings were carried out over a 4-6 week period. After the last day of application 50% of the initial concentration could still be measured in the liquid phase after 1.3 days. The decrease in the concentration followed an exponential function and was attributed to both evaporation and partitioning to sediment. This estimated half-life of 1.3 days for HCB in water compared favourably to that found in the laboratory evaporation tests of Scheunert (unpublished, cited in Schauerte et al., 1982) where 1.7 days (20°C, 1 m water depth) was measured. It should be noted that the 50 µg/L HCB concentration employed by Schauerte et al. (1982) exceeds the measured and calculated solubility of this isomer as given in Table 2. Therefore, it is likely that the measured liquid phase concentration included HCB adsorbed to suspended particulate. No sample filtration was cited in the experimental protocol. It is likely that sedimentation was a major elimination process. About 27% of the total applied HCB was still present in the bottom sediments of the pond after 30 weeks.

Various researchers (Mackay and Wolkoff, 1973; Mackay and Leinonen, 1975; Smith et al., 1981) have developed formulae for predicting the evaporation rates of low solubility chemicals from water. Mackay and Wolkoff (1973) stated that, although compounds having low solubility in water, high molecular weight and low vapour pressure would be expected to evaporate slowly, these compounds often have high activity coefficients in aqueous solution resulting in high equilibrium vapour pressure and, thus, surprisingly high evaporation rates. While chlorinated benzenes were not mentioned speci-

fically, other chlorinated hydrocarbons having low water solubilities were predicted to evaporate rapidly.

Callahan et al. (1979) using published values for vapour pressure and water solubility calculated Henry's Law constants for 5 chlorinated benzenes and compared these to other compounds tabulated by Mackay and Leinonen (1975) for which half-lives had been calculated. It was presumed that the rate of evaporation between compounds having similar vapour pressure and solubility values would be approximately equal. Applying this methodology to all the chlorinated benzene isomers yields half-life estimates for evaporation from a water column 1 metre thick in the order of a half a day or less.

The interpretation of the environmental significance of these calculated evaporative half-lives must be undertaken with care. They assume a one metre water depth and are not directly applicable to shallow or deeper water or where turbulence and mixing are important considerations. More importantly, the calculated half-lives, just as the experimentally determined rates using distilled water, do not take into account adsorption to particulate matter. This phenomenon is discussed in the following section.

1.4.3 Partitioning to Organics

Based on research carried out in the laboratory and in the field, as well as on the environmental levels monitored in the aquatic ecosystem (Section 1.3), it is apparent that chlorinated benzenes tend to partition to organic matter.

Table 9 lists the log K_{oc} values for each isomer calculated by the method of Karickoff et al. (1979) using calculated log K_{ow} values. Log K_{oc}, the logarithm of the organic carbon/water partition coefficient, is a measure of a compound's tendency to partition to organic material (e.g. soil, suspended solids) in the environment. Table 9 indicates that the tendency to partition to organic carbon increases with the degree of chlorination on the benzene ring (Section 1.4.3).

Haas et al. (1974) subjected 1,2,4-TCB to preconditioned treatment plant cultures. The applied concentration was 50,000 µg/L. After 50 hours only a slight decrease in the total TCB concentration was observed. Approximately 40% of the compound was absorbed by the organisms, but was not degraded.

Simmons et al. (1976) also reported the absorption of 1,2,4-TCB to sludge solids. After a 5 day biodegradation using activated sludge, approximately 75% of the TCB which remained in the water was neither biodegraded nor volatilized; it was associated with the waste sludge.

Oliver and Nicol (1982) analyzed the data on raw sewage and treated effluent from four activated sludge wastewater treatment plants in Ontario. Eleven isomers of chlorinated benzenes (MCB excluded) were considered. Although sampling was not extensive enough to adequately define removal efficiencies, crude estimates indicated removals of the chlorinated benzenes were in the 75-95% range. Evaporation and adsorption onto the sludge biomass were suggested as the likely removal mechanisms.

Roberts et al. (1980), studied organic contaminant behaviour during groundwater recharge at a water reclamation research facility in California. Recharge water of known contaminant concentration was injected into a well and the concentrations in the water of an adjacent observation well (8 metres distant) were monitored. For MCB and the three DCB isomers the time to reach the observation well was 36 times and well in excess of 200 times, respectively, of that of a conservative tracer. The concentration response data for MCB and DCB suggested adsorption and not biodegradation was attenuating transport of the isomers. It was presumed that 1,2,4-TCB which was also studied was effectively adsorbed as no detectable concentrations of this isomer reached the observation well by the end of the study period (3 months).

Wilson et al. (1981) studied the transport of MCB, 1,4-DCB and 1,2,4-TCB through 140 cm glass columns packed with sandy soil (0.087% organic carbon). Similar to the findings of Roberts et al. (1980) the higher molecular weight isomers were less mobile and were retained more readily by the soil. Wilson et al. (1981) calculated retardation factors (velocity of tritiated water through soil divided by apparent velocity of the isomer through soil) of 1.7, 3.4 and 9.4 for MCB, DCB and TCB, respectively.

Core drillings at the site of a 1500 gallon spill of Askarel which contained a proprietary solvent of chlorinated benzenes were studied by Moein et al. (1976). The cores indicated that TeCB and QCB were preferentially retained in the soil while other solvent compounds, especially TCB had selectively moved out of the area. Another case study of an Askarel

spill, this one in Saskatchewan, is presented in N.R.C. (1980).

Neely (1980) predicted the distribution of some chemical substances in the various compartments of a simulated ecosystem. The estimated distribution for 1,4-DCB, TCB and HCB is as follows:

Isomer	Water %	Soil %	Air %
1,4-DCB	1	0.3	98.7
TCB	1	4	95
HCB	2	31	67

Although the simplistic model proposed by Neely has been questioned (Zoeteman et al., 1981), it does illustrate the increased partitioning to sediment of the higher molecular weight isomers.

Laska et al. (1976) studied the distribution of HCB in the vicinity of an industrial area along the lower Mississippi River in Louisiana. The mean water concentration of HCB near Baton Rouge was 2.2 µg/L compared to dry weight concentration of 167 µg/kg in the levee soil samples taken beneath the water surface. Laska et al. (1976) attributed the high soil concentrations of HCB to accumulation of HCB from the load carried in suspension and solution in the river.

Laseter et al. (1976) reported high concentrations of HCB were sporadically found in a newly dug pond near an HCB contaminated landfill and in a small stream carrying runoff from a field adjacent to an industrial plant. The HCB concentra-

tions in the pond water varied from 4.8 to 74.9 µg/L and from 10,500 to 53,130 µg/kg in the pond mud. The stream water HCB concentrations varied from 0.1 to 72.8 µg/L and from 2,520 to 13,800 µg/kg in mud samples.

In the experimental pond studies of Schauerte et al. (1982) (described in Section 1.3.3) the residue analyses demonstrated HCB was quickly transported to the bottom sediment. Of the total applied HCB, approximately 27%, 11%, 8% and 7% remained in the bottom sediments after 30, 65, 116 and 145 weeks, respectively. The majority of the HCB in the bottom sediments was located in the upper 10 cm.

In Table 9 the log K_{OC} values calculated by the method of Karickoff et al. (1979) are reproduced for each isomer and compared to those determined from reported concentrations in water and sediment in Lake Ontario, Lake Huron and the Niagara River. The calculated log K_{OC} values are expressed on an organic carbon basis; hence, they cannot be directly compared to the observed values which are based on partitioning to the sediment in its entirety and not the organic carbon fraction alone. If the observed values were corrected for the actual organic carbon fraction present in the sediment, the tabulated observed values could be higher.

As shown in Table 9 the calculated log K_{OC} values are similar to those determined for Lake Ontario and Niagara River sediments. However, since the correction for the organic carbon fraction of the sediment has not been taken into account in the observed values, it is apparent that the calculated log K_{OC} greatly underestimates the potential accumulation possible in the natural environment. At each of

the three locations considered, it appears that the observed log K_{OC} values increase with an increase in the molecular weight of the isomer. This is consistent with the calculated log K_{OC}.

Based on the information presented in this section chlorinated benzenes, particularly the higher molecular weight isomers, can concentrate on particulate matter to levels several orders of magnitude greater than that in the water column. Persistance of these compounds in sediment, therefore, becomes an important environmental consideration. Unfortunately, data on the half-lives of the chlorinated benzene isomers in sediment are limited and are mostly restricted to HCB.

Isensee et al. (1976) observed no HCB loss from soil samples after 12 months storage in covered containers. Soils were treated with 100 to 100,000 µg/kg of HCB and stored under aerobic (sterile and nonsterile) and anaerobic (nonsterile) conditions.

Beall (1975) found a 55% HCB loss from the surface 2 cm of soil after 2 weeks in a simulated pasture experiment. From the 2 to 4 cm soil depth no HCB loss was observed during 19 months. Volatilization appeared to be the removal mechanism operating at the surface layer. Without volatilization losses, HCB would appear to be quite persistant in soil.

Beck and Hansen (1974) in a series of laboratory experiments investigated the half-lives of QCB and HCB in soil incorporated with 10 kg/ha of the compounds. Half-life estimates based on the average of two tests were 0.74 and 4.2 years for QCB and HCB, respectively.

section 2

CHLORINATED BENZENES AND BIOTA

2.0 CHLORINATED BENZENES AND BIOTA

2.1 Levels in Aquatic Organisms

Other than some limited plant and invertebrate data from the lower Mississippi River (Laseter et al., 1976) and from a clam (Elliptio complanatus) bioaccumulation experiment in the Niagara River (Environment Canada/MOE, 1981), all of the information on chlorinated benzene levels in aquatic biota is for fish (Tables 5 and 7).

The observed concentrations are the result of a wide variety of exposure levels and durations which are in turn influenced by a wide variety of other external and internal factors, the sum of which influence the final body burden. Despite the large ranges encountered, the information suggests that a wide variety of chlorinated benzenes are present in a number of fish species captured in diverse areas of North America and abroad. This data is summarized in Table 5 employing broad geographic divisions.

It is not certain whether MCB is rarely detected or, as is more likely the case that it has not been analyzed for (see Section 1.3). Nonetheless the general trend of chlorobenzenes in fish suggests that the more highly chlorinated compounds are present in aquatic biota at concentrations higher than would be expected from their production and use estimates (see Section 1.1). This can be explained by the known properties of chlorinated benzenes - increasing K_B, half-life and persistence with increasing chlorination - that are discussed at length in Sections 1.2, 1.4 and 2.2.

2.2 Accumulation and Elimination by Aquatic Organisms

Bioaccumulation of chlorinated benzenes in aquatic biota occurs whenever the uptake rate exceeds the rate of degradation or elimination. The term bioconcentration is usually used to describe uptake directly from the water through the gills or epithelial tissue. Bioaccumulation is a broader term which includes bioconcentration plus uptake from dietary sources. Biomagnification refers to a process by which tissue concentrations of bioaccumulated substances increase as they pass up the food chain through two or more trophic levels (Macek et al., 1979).

Bioaccumulation factors (K_B) are defined as the mean equilibrium body burden of a particular chemical divided by the mean exposure concentration to which the species was subjected. These factors can be estimated by either direct measurement of body burden and exposure water concentration or by determining the ratio of the kinetic rate constants k_1 and k_2 which describe the uptake and clearance of the chemical from the organism, respectively. This latter method is described by Branson et al. (1974) and has been applied by the Aquatic Toxicity Research Group (unpublished, a,b,c) and Neely et al. (1974).

The ability of aquatic organisms to bioaccumulate chlorinated benzenes in their tissues to levels in excess of those to which they have been exposed in the aquatic environment has been demonstrated by numerous investigators. As discussed in Section 2.1 this phenomena is illustrated via the field data shown in Tables 3 and 5 and, as well, in Table 7. Table 10 summarizes bioconcentration factors (K_B) for some aquatic species determined in various controlled studies.

2.2.1 Review of Bioconcentration Studies

As can be seen in Table 10 the K_B values tend to increase for the higher molecular weight isomers. This has been demonstrated in studies on: rainbow trout (Salmo gairdneri) by Neely et al. (1974), Veith et al. (1979a) and Oliver and Niimi (1983); bluegill sunfish (Lepomis macrochirus) by U.S. EPA (1978a); green sunfish (Lepomis cyanellus) and fathead minnows (Pimephales promelas) by Veith et al. (1979a); guppy (Poecilia reticulata) by Konemann and van Leeuwen (1980); and flagfish (Jordanella floridae) by Aquatic Toxicity Research Group (unpublished, a,b,c).

Comparison between K_B values determined for the same isomers by different researchers shows that there is considerable variation in the values reported in the literature. These ranges, which are derived from Table 10, and also appear in Table 9, are listed below expressed as log K_B on a whole body basis.

Isomer	Log K_B (whole body)
MCB	2.81 - 3.62
1,2-DCB	1.95 - 2.74
1,3-DCB	1.82 - 2.87
1,4-DCB	1.78 - 2.86
1,2,3-TCB	2.85 - 3.51
1,2,4-TCB	2.26 - 3.37
1,3,5-TCB	2.88 - 3.61
1,2,3,4-TeCB	3.72 - 4.08
1,2,3,5-TeCB	3.26 - 3.59
1,2,4,5-TeCB	2.74 - 4.11
QCB	3.53 - 4.30
HCB	1.20 - 4.65

The observed variations in the reported values can be attributed to the use of different test species, exposure concentrations, exposure durations and experimental protocols.

Model Ecosystem Studies

Lu and Metcalf (1975) studied the bioaccumulation of MCB and HCB in green filamentous alga (Oedogonium cardiacum), snails (Physa sp.), water fleas (Daphnia magna), mosquito larvae (Culex quinquifasciatus) and mosquito fish (Gambusia affinis). For both MCB and HCB the observed K_B values were greatest for the algae with measured accumulations of 4185 times and 3969 times, respectively. Corresponding increases in the MCB and HCB body burden in the mosquito fish were 645 times and 1166 times, respectively.

In the experimental aquatic ecosystem of Metcalf et al. (1973) the snail accumulated the greatest body burden equivalent to 1248 times the HCB exposure concentrations. Algae accumulated 522 times and fish 287 times the exposure concentration.

The bioaccumulation potential of HCB in algae (K_B of 610 to 910) reported in the aquatic ecosystem studies of Isensee et al. (1976) are in better agreement with those reported by Metcalf et al. (1973) than with those reported by Lu and Metcalf (1975). However, unlike the two latter investigations, Isensee et al. (1976) found HCB tended to accumulate in higher food chain organisms. Mosquito fish and snails (Helisoma sp.) always contained 1.5 to 2 times more HCB than the algae and daphnids. Catfish (Ictalurus punctatus) accumulated approximately 10 times more than did the other organisms.

Niimi and Cho (1980) have suggested that higher trophic level organisms would be more likely to accumulate HCB. Their studies showed fish fed diets containing HCB attained body

burdens related to the concentration in the food and the duration of feeding.

2.2.2 Limitations of Current Bioconcentration Information

The tendency for chlorinated benzenes to bioaccumulate in higher food chain organisms cannot be quantified with the K_B data in Table 10. Aside from the aquatic ecosystem investigations presented above, most K_B studies of chlorinated benzenes have been restricted to fish species. Even between different fish species there is considerable variation in bioaccumulation reported by investigators. For example, Laseter et al. (1976) observed HCB to bioaccumulate to 44,440 times in largemouth bass (Micropterus salmoides) and only 3578 times in sunfish (Lepomis macrochirus). In both cases it must be noted that steady-state conditions had not been established during the uptake phase of the experiment. Similarly, the Aquatic Toxicity Research Group (1982, unpublished c) reported 1,2,4,5-TeCB to bioaccumulate to 555 times in brook trout (Salvelinus fontinalis) and 4050 times in flagfish (Jordanella floridae). The difference in lipid content of the two test species (2.6 and 8.5%, respectively) could not account for all of the variation in the reported K_B values.

Exposure Concentration

The effect of exposure concentration on the degree of bioaccumulation has been reported by some researchers. Isensee et al. (1976) reported the amounts of HCB accumulated by the five species tested were directly related to exposure concentrations; the higher the exposure concentration, the greater the observed accumulation and K_B. Laseter et al. (1976)

also reported that the quantity of HCB accumulated in large-mouth bass tended to increase in relation to the exposure concentration; however, the K_B values calculated using these observed body burdens yielded slightly higher K_B values for the lower concentrations tested.

Oliver and Niimi (1983) reported that at the higher exposure concentrations used in their investigations, the K_B values averaged 2.2 times higher than at the lower exposure concentrations tested for each of the ten chlorinated isomers investigated. In this study, the low exposure concentrations were near environmental levels, the high exposure concentrations were about an order of magnitude greater. It was suggested that the increase in K_B associated with the higher dose may indicate that the rates of detoxification and elimination of these chemicals by the fish are concentration-dependent.

Another factor which may be contributing substantially to the variability seen in bioaccumulation estimates for the same compound is the bioavailability of the test compound concentration to the exposed organisms. For a given nominal concentration the particulate and dissolved fractions may be differentially available for uptake (ATRG, unpublished d). Even the dissolved portion of some hydrophobic organic compounds (polyaromatic hydrocarbons, PAH) can be divided into two phases - a portion sorbed to dissolved humic matter and a portion not - which are differentially available (McCarthy and Jiminez, 1983). Differing bioavailability related to partitioning to differentially available dissolved and particulate phases represents a significant potential confounding factor for which there is little detailed information.

Exposure Duration

The duration of exposure and the experimental methodology employed (i.e. continuous versus static flow systems) are also partially responsible for the variation in K_B values reported for the same isomer in Table 10. In many of the investigations summarized in Table 10 it is uncertain whether the study durations were sufficiently long to ensure that steady state conditions were reached. As stated previously this was clearly a problem with the research of Laseter et al. (1976). Their data showed that HCB residues were continually increasing in all 5 test species even after 15 days exposure. Therefore the K_B determined would not be representative of equilibrium conditions.

Similarily, in another phase (not shown in Table 10 since water concentrations were not measured) of the investigation of Isensee et al. (1976) catfish exposed to three different levels of HCB contaminated soil continued to steadily accumulate HCB over the 20 day duration of the test. Therefore it is probable that the K_B of 15 840 times calculated by Isensee et al. (1976) for catfish after an eight day exposure (Table 9) is conservative.

In the studies of Macek et al. (1979), 1,2,4-TCB residue concentrations in daphnia (Daphnia magna) and bluegill sunfish (Lepomis macrochirus) were measured throughout the 28 day duration of the experiment. Steady state residue concentrations of 1,2,4-TCB in daphnia were achieved in 4-8 hours and within 3 days in the bluegill sunfish as a result of aqueous exposure, and within 14 days as a result of dietary exposure.

Oliver and Niimi (1983) reported that rainbow trout (Salmo gairdneri) attained equilibrium concentrations within 7 days for 9 of the 10 chlorinated benzenes tested at a high exposure level. The exception was HCB which did not reach equilibrium in the fish tissue after 105 days. At the low level of exposure, equilibrium was attained for the DCB and TCB within 8 days, while 1,2,4,5-TeCB required at least 39 days and 1,2,3,4-TeCB and QCB at least 69 days. Again HCB did not reach equilibrium within the duration of the 119 day test.

As the investigations presented above suggest, the determination of steady state K_B values requires that the tests be undertaken over a sufficiently long period. This is particularly true for the more highly chlorinated compounds. However, as the studies of Lu and Metcalf (1975) indicate, significant quantities of these compounds can be bioconcentrated in a very short period of only one to two days. It is not certain though whether this represents internal uptake or external accumulation on mucous and/or gills.

The static system used by Lu and Metcalf (1975) also makes the K_B determined by these investigators suspect, particularly for MCB. Approximately 96% of the applied MCB was retained in their volatilization trap. The water concentration of MCB used to determine the K_B was that measured at the termination of the experiment, about 4% of the original exposure concentration. This value was reached only at the end of the experiment and is not representative of the actual exposure to MCB which was received by the organisms. This ranged from 100% of the MCB at the start of the experiment through to the 4% which was observed at the end. If an integrated exposure value, reflecting the decreasing water concentration over the length of the experiment, was used,

the K_B values obtained for MCB would be less. Unfortunately, no other research is available to verify or quantify this.

Dietary Contribution to Body Burden

In the ecological model studies of Metcalf et al. (1973), Lu and Metcalf (1975) and Isensee et al. (1976) described earlier, the contribution of aqueous and dietary sources to the measured body burden were not quantified. Therefore it is not possible to determine whether the K_B values calculated are measures of bioconcentration or bioaccumulation. Macek et al. (1979) tested the relative contributions of dietary and aqueous exposure of bluegill sunfish (Lepomis macrochirus) to 1,2,4-TCB. Accumulation attributed to dietary sources was calculated as only 6% of the total body burden. This was determined to be statistically insignificant when compared to the error associated with the determination of body burden due to aqueous exposure alone. Employing the same test species, but using HCB, Laseter et al. (1976) found that after seven days the dietary contribution of HCB was approximately 17% of that from aqueous exposure alone.

The findings of Macek et al. (1976) and Laseter et al. (1976), suggesting that the dietary contribution of chlorinated benzenes to the total body burden of fish is small, is not borne out in the studies of Niimi and Cho (1980, 1981). Laboratory feeding studies indicated that the uptake of HCB from contaminated food was directly related to the concentration in the food and the duration of feeding. Also these investigators reported that field observations of HCB residues in Lake Ontario salmonids were much greater than what laboratory K_B values and the low HCB concentrations in the

lake would indicate. Therefore, in natural waters where waterborne HCB levels are extremely low, uptake from food could be substantially greater than that from water (Niimi and Cho, 1981).

2.2.3 Other Bioconcentration Factor Information

In addition to the experimental evidence cited above, there is other evidence that the bioaccumulative potential of the chlorinated benzenes increases with increased chlorination. Many researchers (Neely et al., 1974; Lu and Metcalf, 1975; Veith et al., 1979a; Konemann and van Leeuwen, 1980; Mackay, 1982; and Oliver and Niimi, 1983) have demonstrated a correlation between log K_B and log K_{ow} (the logarithm of the octanol/water partition coefficient) for a wide variety of hydrophobic chemicals. Log K_{ow} is a commonly used indicator of the lipophilic nature of chemicals and as the empirical correlations suggest, the greater the log K_{ow} the greater the bioaccumulative potential.

Using the calculated log K_{ow} values from Table 2 and the one-constant correlation equation derived by Mackay (1982), the K_B for each chlorinated benzene compound was calculated and is presented in Table 9. For comparison, the range of experimentally determined log K_B from Table 10 are also provided in Table 9.

Table 9 demonstrates that there is generally close agreement between the experimentally and the empirically derived log K_B, except for MCB and HCB. The experimentally determined log K_B range for MCB is from the research of Lu and Metcalf (1975). As previously discussed, Lu and Metcalf (1975) employed a static flow system and experienced volatilization

losses in the order of 96%. The exposure concentration used in the calculation of the K_B values was that of a measurement taken at the end of the study. This measurement was not representative of the actual concentrations to which the test species were exposed; hence, the K_B values calculated were probably high. The lack of agreement between the experimental and empirical log K_B for HCB can probably be attributed to the long time required to reach steady-state HCB body burdens (Oliver and Niimi, 1983) and the typically short duration of the laboratory uptake studies.

In Table 9 log K_B values have been calculated for fish in Lakes Ontario and Huron and the Nigara River based on field data of water and fish residue concentrations (Environment Canada/MOE, 1981; Oliver and Nicol, 1982). The log K_B calculated for these natural waters often exceed those derived in the laboratory or those predicted empirically. This is consistent with the findings of Niimi and Cho (1981) who suggested the levels of HCB monitored in fish from natural waters where waterborne levels are extremely low, may be due to substantial dietary uptake. Oliver and Niimi (1983) attributed higher K_B of HCB in fish of natural waters as compared to those in laboratory studies, to the long half-life of HCB in fish. If the rate of intake exceeds the capacity of the fish to eliminate HCB the body burden of HCB would be expected to continually rise.

2.2.4 Metabolic Breakdown and Elimination

The current understanding of the metabolism of chlorinated benzenes is incomplete; however, some general trends are evident. All animals studied to date, both aquatic organisms (as noted above) and standard laboratory test animals (mainly

mammals, see U.S. EPA, 1980a,b) appear to use a similar metabolic pathway. This is evidenced by the similarity of the major metabolites, the chlorophenols. The basic approach is to form a more polar metabolite to facilitate excretion. The lower chlorinated benzenes appear to undergo a hydroxylation reaction while the higher ones undergo a hydroxylation-dechlorination reaction (Safe et al., 1976; Sanborn et al., 1977; U.S. EPA, 1980a,b). In these processes highly toxic arene oxides may be formed as intermediates (Kohli et al., 1976).

The more highly chlorinated compounds are generally more resistent to metabolic breakdown, and although rates appear to vary widely between organisms, the relative order - MCB being metabolized the fastest; HCB the slowest - is maintained. Safe et al. (1976) have suggested that amphibians (frogs) exhibit metabolic breakdown rates that appear to be faster than fish (trout) but slower than mammals. Generally, the rate of metabolic breakdown and elimination of chlorinated benzenes from biota decreases as the molecular weight of the compound increases.

Metabolic Breakdown

The metabolic breakdown of chlorinated benzenes in the aquatic biota has been studied by Lu and Metcalf (1975), Safe et al. (1976), and Sanborn et al. (1977).

The most extensive of these studies was that of Safe et al. (1976) who investigated ten of the twelve chlorinated benzenes. They injected frogs (Rana pipiens) with chlorinated benzenes in a vegetable oil carrier and examined the water in which the frogs lived for metabolites. The amounts and types

of metabolites in the body were not investigated. One per cent or less of the initial doses was converted to a metabolite and excreted into the water over the 8 day period. Specifically for MCB, DCB, TCB and TeCB the amounts of metabolites excreted were 1%, not reported (probably 1%), about 0.7 to 1%, and less than 1% respectively. No metabolites of QCB or HCB were detected in the water. The metabolites of MCB, DCB and TCB were chlorophenols that are direct hydroxylation products while the TeCB isomers yielded chlorophenols that are typically the result of hydroxylation-dechlorination reactions.

Lu and Metcalf (1976) studied the metabolic products found in algae, daphnia, mosquitos, snails and fish after 2 or 1 day exposures to MCB and HCB. For MCB the identified metabolites represented about 15%, 24%, 97%, 58% and 75%, respectively, of the amount of MCB found in the body at the time. For HCB the values were about 6%, 47%, 7%, 0% and 0%, respectively. The identified metabolites were ortho-and para-chlorophenol and 4-chlorocatechol for MCB and pentachlorophenol for HCB. Generally a larger variety of unidentified metabolites was reported for HCB, although for both compounds this varied with the organism in question. Although the metabolites are similar to those reported by Safe et al. (1976), the rates are not, as no measurement of the amounts excreted into the water was made. Sanborn et al. (1977) also reported that in fish pentachlorophenol was the major metabolite, although several other unidentified ones were reported.

A very important observation with respect to the metabolic handling of chlorinated benzenes is the observation that pre-exposure appears to reduce the subsequent rates of depuration, when compared with the rates from non pre-exposed

fish (ATRG a, unpublished). Among other things this could explain the higher K_B values reported in long term studies (Oliver and Niimi, 1983) and field data (Table 9) versus short-term studies (see Table 10). It could also explain the observation that chlorinated benzenes have low acute toxicities yet a variety of serious subacute and chronic effects have been noted from extended exposures to relatively low concentrations (U.S. EPA, 1980a,b; Ware and West, 1977).

Elimination

Neely (1979, 1980) developed a relationship to estimate the rate of clearance of chemical compounds from biota. Half-lives of clearance were found to be a function of a chemical's molecular weight, solubility and vapour pressure. Estimates of half-life in biota have been calculated for each isomer according to the method of Neely (1979) using molecular weights, vapour pressures and calculated solubilities from Table 2. These calculated half-life estimates are shown in Table 8 versus observed depuration half-lives determined in laboratory studies. Although the observed half-lives tend to be higher, both the calculated and observed half-lives illustrate the increased persistance of the higher molecular weight compounds in aquatic organisms.

Sanborn et al. (1977) studied the elimination of HCB by green sunfish (Lepomis cyanellus). These investigators reported the rate of removal of HCB differed from various organs and tissues. While the depuration half-life for the whole carcass was estimated as 10 days, half-lives of 20 days and about 8 days were estimated for the liver and other tissues (stomach, intestine and muscle), respectively.

2.3 Toxicity to Aquatic Organisms

In any investigation of the effect of pollutants on water quality the impacts, both actual and potential, on aquatic organisms are of major concern. Since they reside in the water and are exposed to pollutants for virtually their entire lifetime, albeit at varying concentrations, they are often the most sensitive indicators of adverse conditions. Although the impact of pollutants on humans is clearly a major concern, in many cases establishing levels that provide protection for aquatic organisms achieves a considerable measure of protection for humans. As well, although acute toxicity information for mammals is usually at least as abundant as that for aquatic organisms, there is often more longer-term chronic test information for aquatic organisms due to the lower cost of longer-term tests with aquatic organisms. Chronic tests are also more useful when attempting to assess the impact and determine "safe" levels for long-term, low exposure conditions that are more typical of the real-world situation.

A considerable amount of aquatic toxicity data is currently available, as can be seen in Tables 11 to 14; however, all of it is not of equal utility in setting water quality guidelines. As will be discussed later, there are various technical reasons for these difficulties. As well, mammalian toxicity information, although of use in a general way, is difficult to interpolate to the aquatic environment and its inhabitants. As will be seen later, the primary data base which remains is that for fish. However, prior to further discussion of this, four points must be addressed: chemical purity, expression of exposure concentration, water solubility, and bioavailability.

The first point to be dealt with is the matter of the purity of the compounds being tested. Although researchers sometimes note the source (manufacturer or distributor) of a particular compound and the particular purity grade in question, details of the purity and major contaminants are not routinely given. Based on information given in Section 1.0 it appears that both compound and isomer cross-contamination is virtually certain, although the extent is not quantified (Ware and West, 1977). Although no information on contamination by related compounds, such as chlorophenols, is readily available, it is very likely that small amounts of these materials are also present. The impact of contamination is not known but should not be ignored as, among other things, it may be a significant source of inter-test variability.

Secondly, most authors have reported their exposure concentrations as a weight per litre of water (i.e. µg/L) and Tables 11 to 14 follow this procedure. The data discussed in Section 4.0 have been converted to molarity (µMoles/L). Molarity values are obtained by dividing the weight of the compound in question by its known molecular weight (see Table 2). In discussions of toxicity, especially when comparisons of different molecular weight compounds are being made, molarity values are much more indicative of the relative toxicity of the various materials. It is commonly acknowledged that in toxicological and pharmacological studies toxicity is typically a result of the number of molecules of a toxicant present, not the weight. The value of this procedure, especially for this group of related chemicals, will become more apparent in Section 4.0.

Thirdly, one of the major difficulties in obtaining accurate information with standard bioassays results from the fact

that the water solubilities for the various chlorinated benzenes range from low to extremely low (see Table 2). The simplistic approach of reporting the results of a study on the basis of an unmeasured nominal concentration can create a significant error that increases with the higher molecular weight compounds.

This is a problem for both static and flow-through tests. For example, Konemann and van Leeuwen (1980) report that in their flow-through experiments the measured concentrations ranged from 73 to 8 per cent of the nominal concentration for a series of chlorinated benzenes ranging from 1,4-DCB to HCB, respectively. ATRG (unpublished, b, c) also report on this phenomenon with 1,4-DCB being 17 per cent of the nominal and 1,2,4-TCB being 20 per cent of the nominal concentration used. In addition, they noted that at lower nominal concentrations the measured concentration is closer to the nominal value than is the case at higher nominal concentrations.

Various methods have been used in an attempt to increase the solubility. The approaches fall into two basic categories: employment of a solvent or carrier material such as acetone (ATRG, unpublished a-c; Buccafusco et al., 1981; LeBlanc, 1980) or continuous mechanical agitation (Birge et al., 1979; Black et al., 1982). The effectiveness of these procedures is not well documented relative to the unmodified state and the influence of solvents on the bioavailability of the compounds has not been established experimentally. It is likely that both procedures increase bioavailability, thereby making test results not directly comparable with standard procedures.

The fourth and last point to be discussed is bioavailability. Undissolved or precipitated material has been noted in exposure tanks with and without the presence of solvents or carriers and in both static and flow through tests (Buccafusco et al., 1981; ATRG, unpublished, b,c,d). Observations by the ATRG (unpublished d) outline this problem. A 96 hr LC_{50} for 1,2,4,5-TeCB was determined to be about 2000 µg/L; however, when finely divided particulate matter was removed from the water sample either by filtration (0.2 to 50 µm pore size extrapolated back to a 0 µm pore size) or centrifugation it was discovered that the actual content of the water was about 400-500 µg/L, the estimated maximum solubility of the compound in water. At a measured concentration in water of 500 µg/L no mortality was noted in a 96 hour exposure. Although it is likely that both the dissolved and particulate are contributing to toxicity, it is not clear whether they are equally bioavailable.

Further to this are the observations of McCarthy and Jimenez (1983). Working with the hydrophobic organic compounds, polyaromatic hydrocarbons - PAH - they reported that the waterborne dissolved phase of hydrophobic organics can be further divided into two phases, that sorbed to humic matter dissolved in the water and that not sorbed. By varying the humic content dissolved in the water, they were able to establish that sorbed PAH is substantially less available than the non-sorbed PAH. Since the humic matter content - representing a major portion of the dissolved organic phase in the water column - will vary in space and time in the ecosystem as well as at various laboratory test facilities, and yet is not routinely reported, it may be introducing a substantial amount of variability to the expression of toxic effects. This may explain a considerable portion of variability reported for otherwise similar toxicity tests.

Also important in the question of bioavailability is the fact that the effect may be increasingly more pronounced as hydrophobicity rises. The organic carbon partition coefficients (log K_{oc}, see Table 2) indicate that as the hydrophobicity (decreased water solubility and increased log K_{oc}) increases, the partitioning to the organic phase increases. That is to say, the more highly chlorinated of these compounds are more likely to have a higher proportion of the total water column load present in the organic phase -both as undissolved material sorbed to fine particulate matter and as dissolved material associated with organic ligands - than the less chlorinated ones.

It is apparent that the bioavailability of chlorinated benzenes, and the subsequent effect on toxicity, although not yet well-understood, is a function of at least two factors. The first is the amount of organic material present in the water. This is a reflection of the capacity of the organic phase to contain hydrophobic compounds. It may be subdivided into particulate and dissolved phases. The second is the degree of hydrophobicity, as measured by K_{ow} or K_{oc}. This reflects both the water solubility and the tendency of the compound present in the water to partition into the organic phase.

2.3.1 Mode of Toxic Action

In general, the mode of toxic action of the chlorinated benzenes is not well understood. In fish the symptoms of acute intoxication by various chlorinated benzenes, varying from MCB to QCB, has been reported to result in convulsions, erratic swimming behaviour, and loss of equilibrium; all of which

are indicative of an generalized effect on the central nervous system known as narcosis (Dalich et al., 1982; Smith, 1981; Verth et al., 1983). This is consistent with the anesthetic effects of chlorinated benzenes reported in laboratory studies with mammals (U.S. EPA, 1980 a,b).

Information on the modality of sublethal and chronic toxicity in fish is limited to MCB and HCB. Both of these compounds were reported to be hepatotoxic on the basis of histological and biochemical evidence (Dalich et al., 1982; Laseter et al., 1976). HCB also appeared, on the basis of histological evidence, to affect the kidney and gall bladder (Laseter et al., 1976). Chlorinated benzenes have been reported to be hepatotoxic in studies with laboratory mammals (U.S. EPA, 1980 a,b).

The metabolism of chlorinated benzenes is discussed in more detail in Section 2.2. However, one point should be mentioned here. If, as suggested by Safe et al. (1976), fish, amphibians and mammals metabolise chlorinated benzenes in a similar manner, albeit at different rates - fish slowest, mammals fastest; MCB fastest, HCB slowest-, then the metabolic pathway postulated for breakdown to the major metabolites, largely chlorophenols, may be important in this matter. Kohli et al. (1976) suggest that the highly toxic arene oxide intermediates formed in the metabolism of most chlorinated benzenes may, at least in part, be the source of the toxicity of these compounds.

The influence of the characteristics of the exposure water in toxicity is limited to the observation by Birge et al. (1979) that the toxicity of MCB to largemouth bass was similar in waters of 50 and 200 ppm hardness.

2.3.2 Acute Toxicity

Acute toxicity data falls into two major categories: (i) lethal concentration (LC) information, based on a water exposure, and (ii) lethal dosage (LD) information based on an amount introduced into the body of the animal via the digestive system or by direct injection. The former information will be discussed first.

The presently available lethal concentration data for freshwater studies are presented in Table 11. These data can be divided into the two test methodologies employed: static tests and flow through tests. Many reported test results, especially from static tests, are often based on unmeasured, nominal exposure concentrations, a practice that, as discussed earlier, is demonstrably unsound. Therefore, for the purposes of this report, test results based on nominal concentration estimates will not be used any further than to say that these data suggest that the higher molecular weight chlorinated benzenes appear to be more acutely toxic.

Discussions subsequent to this point deal, for the most part, with data where the concentrations have been measured; primarily flow-through tests. Within this data set a wide variety of experiments carried out using a number of different test organisms and exposure regimes, have been conducted. Although 96 hours is the generally accepted standard exposure duration it can be seen that a variety of other exposure times, some longer some shorter, have been employed. Although the variability from this source, as well as that resulting from different researchers, different species and the different toxicity of the various isomers of

any particular compound, is readily apparent, a general trend to increasing toxicity with increasing chlorination and hydrophobicity is apparent. This data will be discussed further in Section 4.0.

No saltwater data have been included in this report although the static unmeasured information reported by Heitmuller et al. (1980) suggests a trend similar to that noted above.

Table 12 contains some information on the relative toxicity the chlorinated benzenes based on 96 hour LD_{50} data (intraperitoneal injection in an oil carrier) for rainbow trout (Kaiser et al., in press). Smith (1981), on the basis of an injection study with rainbow trout, reports LD_{50} values of 54.5 and 14.8 and ED50 (estimated lethal dose) of 10.6 and 3.41 mMoles/kg for 1,2-DCB, 1,2,4-TCB, 1,2,4,5-TeCB, and QCB, respectively. Dalich et al (1982) in a similar study reports a 96 hour LD_{50} of 9.8 mMoles/kg for MCB. Laseter et al. (1976) reports some mortality in crayfish over a two month period at doses of 0.06 to 0.44 mMoles/kg of HCB in a peanut oil carrier although no LD_{50} information is derived. They also reported no mortality over a one month period when 0.44 mMoles/kg of HCB was injected, in a peanut oil carrier, into gulf killifish (Fundulus grandi), a saltwater species.

LD_{50} data for other organisms is readily available but less comparible than that presented by Kaiser et al. (in press) for fish. Results based on a variety of LD_{50} tests, both oral and injection studies, with laboratory animals (rats, mice, guinea pigs, and rabbits) generates LD_{50} values of 23-37, 13-24, 17-50, 4.2, 4.6-12 and >3.5 - >10.6 for MCB, 1,2-DCB, 1,4-DCB, 1,2,4-TCB, 1,2,4,5-TeCB and HCB, respectively (U.S. EPA, 1980 a, b).

All of the LD_{50} data mentioned above are important for interpretation of the physiological explanations of chlorinated benzene toxicity, but for the first order approximation, the toxicity information developed in aquatic systems must take first priority.

Two points developed in the LD_{50} research are worthy of note here as they may have importance in interpreting aquatic toxicity information. Doses of chlorinated benzenes appear to be cumulative, i.e. the equivalent of a lethal dose given in a series of sub-lethal doses will cause the same effect in laboratory animals. This has been observed for 1,4-DCB and 1,2,4,5-TeCB. This is supported by the observation that, although chlorinated benzenes have been observed to have a low acute toxicity, a variety of toxic effects have been noted from prolonged exposure to comparatively low levels (U.S. EPA, 1980 a,b; Ware and West, 1977).

A very important point from LD investigations with fish is that the toxicity of a wide variety of substituted benzenes (xylene, toluene, phenols, chlorinated benzenes) and benzene itself was simply additive (Smith and Craig, unpublished). Data collected on mixtures of 1,4-DCB and 1,3-4-TCB during LC_{50} investigations with fish also support this observation (ATRG, unpublished, a).

2.3.3 Chronic Toxicity

The data on algae presented in Table 14 are not readily comparable with the fish data for two reasons: (i) it is difficult to compare between the different measures of chronic toxicity utilized in fish and algal studies, i.e. is inhibition of fish growth equivalent to inhibition of algae growth,

and (ii) the algae data are generated in static tests employing a nominal unmeasured toxicant concentration. Although the data on algae support the trend observed elsewhere, that of increasing toxicity with increasing molecular weight, this information will not be considered any further for these reasons.

The chronic data on animals, which appears in Table 13, consists of results from two related types of tests: (i) the early life stage (ELS) test, which attempts to derive a threshold level for chronic toxicity effects based on growth, survival and hatchability, and (ii) long duration 7.5 to 27 day LC_{50} estimates using embryo and larval fish (embryo larval or E-L test).

The data for MCB, the only chronic data available for aquatic animals other than fish, suggest that fish are more sensitive than amphibians (Black et al., 1982). The chronic estimate for HCB, based on histological evidence, is not readily comparable with other test results. A 12 day LC_{50} result for QCB (U.S. EPA, 1980; appears in Table 10), an incomplete ELS test for 1,2,4,5-TeCB (ATRG, unpublished d) and a 14 day LC_{50} estimate (Calamari et al., 1982) are examples of other non-standard indications of chronic toxicity.

The chronic data on MCB were generated by the same group (Black et al., 1982 and Birge et al., 1979) using a modified flow-through test apparatus. Constant mechanical agitation was employed and volatility was controlled by using an exposure chamber with no air-water interface. These data may not be readily comparable with all of the other information which was produced using an unmodified standard technique.

The only case in which a toxic effect of chlorinated benzene is associated with a measured tissue or organ level is for HCB. Laseter et al. (1976) reported that liver and kidney damage was evident at HCB tissue loads of about 3000-28,000 µg/kg. These loads resulted from the exposure of bass (Micropterus salmoides) to HCB levels of about 3.5 µg/L over 10 or 15 days. The damage was detected on the basis of histological information. It is not known if this damage can cause serious debilitation of the fish (no gross effects were reported in the study) and there is no way to relate this to any of the other toxicological data; however, it is the only data of this nature available.

2.4 Wildlife

There is little information on which to base an assessment of the effects of chlorinated benzenes on wildlife. The information currently available is largely on birds. These data are presented in Table 6.

Additionally, Koss and Manz (1976) report on HCB levels foxes (Vulpes vulpes), wild boars (Sus scrofa) and deer (Capreolus capreolus) in West Germany. Mean HCB levels in the subcutaneous adipose tissue were 260, 710, and 30 µg/kg, respectively. The authors suggest that the wide difference in levels between the deer (a vegetarian) and the fox and boar (predators of small mammals and invertebrates) indicate that accumulation via the food chain is occurring. The data reported in Table 6 support the contention that considerable accumulation is occurring in birds because the tissue levels of various chlorinated benzenes are clearly very much higher than typical water concentrations (see Table 3).

In laboratory studies, birds (chickens) have been shown to accumulate HCB (Avrahami and Steele, 1972). Japanese Quail (Coturnix coturnix) - subjected to high doses in food - up to 80 mg/kg - have been shown to exhibit reduced egg production and reduced fertility and hatchability. A no-effect level was estimated to be 1 mg/kg in the diet (Vos et al., 1971). It does suggest that, like mercury and PCBO (MOE, 1979), there is a body burden that will cause reduced reproductive success and other chronic effects and that a water concentration criteria can be formulated, knowing the properties of the compounds, such that accumulation to this undesirable level is precluded. This is especially important for birds and mammals whose diet is largely composed of fish.

If 1 mg/kg of HCB in the diet is of the correct order of magnitude to provide protection for these organisms then it is in reasonable agreement with suggested limitations for human foodstuffs reported in Section 4.0.

Concentrations of HCB in fish in the Great Lakes are below the 1 mg/kg level (Table 5), as are the levels in birds (Table 6). This suggests that current levels of HCB in Great Lakes water may not be problematic.

However, this data is rather limited in nature and it would be premature to exploit it for rigorous use in wildlife criteria formulation. It must be concluded that, at this time there is insufficient data on which to base water concentration objectives for chlorinated benzenes which would protect wildlife.

section 3

RECREATION AND AESTHETIC CONSIDERATIONS

3.0 RECREATION AND AESTHETIC CONSIDERATIONS

It is generally accepted that potable water in recreational areas should comply with the levels suggested for raw public water supplies (Reeder, 1979). Although no federal or provincial guidelines for chlorinated benzenes in drinking water exist as of yet, the criteria formulated by other jurisdictions (Table 19) might well be used as an interim measure. Comparison of these criteria with observed water concentrations (Table 3) indicates that all recorded water concentrations in Canada, other than a few industrial discharges, are below the criteria levels cited in Table 19.

The unattractive aesthetic aspects of chlorinated benzenes in water appear to be limited to the possibility of taste/odour problems. The taste/odour thresholds for various chlorinated benzene compounds are listed in Table 15. When these threshold levels are compared against concentrations of chlorinated benzenes observed in Canadian waters (Table 3) it is apparent that, currently, taste/odour do not appear to be a problem, at least with respect to water.

One aspect of aesthetics, however, remains unknown: tainting of the flesh of aquatic organisms. As can be seen by comparing Tables 15 and 5, fish tissue levels of some of the chlorinated benzenes approach or exceed, as a result of bioaccumulation, concentrations where taste/odour problems are observed in water. Although it is not known exactly at what concentration level tainting problems begin to occur, the levels are likely similar to that for water. Tainting of animal tissue does occurs as tainting of eggs and pork meat, resulting from exposure of the animals to 1,4-DCB vapours, has been reported (Jori et al., 1982).

section 4

DEVELOPMENT OF WATER QUALITY OBJECTIVES
FOR THE PROTECTION OF AQUATIC LIFE

4.0 DEVELOPMENT OF WATER QUALITY OBJECTIVES FOR THE PROTECTION OF AQUATIC LIFE

4.1 Introduction

Prior to entering into the formulation of suggested Provincial Water Quality Objectives a summary of the information and discussions made earlier in the report is in order. This will aid in clarifying the approach and understanding the overall rationale that will be employed in guideline formulation.

The chlorinated benzenes are stable chemically and very resistant to abiotic and biotic degradation, with the more highly chlorinated compounds becoming increasingly more resistant to both forms of breakdown. The environmental fate of members of the chlorinated benzene group changes as a function of their respective partition coefficients. The lower molecular weight compounds have low partition coefficients and tend to evaporate to the atmosphere. The higher molecular weight compounds shift more and more towards partitioning and accumulation in the organic component of the aquatic environment. This partitioning to organics is by no means exclusive to the non-living particulate organic matter in the water column and in the sediments. The data on living organisms suggests that bioaccumulation also increases substantially as the degree of chlorination increases.

In addition to being bioaccumulated to a greater extent and being metabolized more slowly, observations indicate that the higher molecular weight compounds are excreted/depurated more slowly. The net result of this is that the more highly chlorinated benzenes have increasingly longer half-lives in aquatic biota.

Although a great deal is not known about the mode of toxicity of this group of compounds, doses of any particular compound appear to produce similar effects, whether introduced as a single dose or a series of lower doses the sum of which is equivalent to the single dose. In addition, the toxicities of the various chlorinated benzenes appear to be simply additive with each other. Symptoms of acute intoxication are similar for all of the compounds so far reported on: loss of equilibrium and erratic swimming activity. This has been attributed to the depression of central nervous system activity, often termed narcosis. Histological and biochemical data suggest the liver, kidney, and perhaps the gall bladder are also involved.

Although little is known about the mechanisms of toxicity, members of the group seem to be handled in a similar way, albeit at differing rates, producing similar metabolites. The less chlorinated isomers undergo a hydroxylation reaction while the more highly chlorinated ones undergo a hydroxylation-dechlorination reaction.

All of the above information leads to the statement of three important considerations:

i) The toxicity of the chlorinated benzenes to aquatic organisms, as measured by typically used tests, increases with the degree of chlorination.

ii) The mode of toxic action seems to be similar within the group, and some evidence suggests the toxic effects may be simply additive.

iii) Differences in a variety of chemical/physical properties, in addition to toxicity, can readily be explained as being a function of the degree of chlorination. This is evidently a closely related group of compounds.

4.2 Development of Objectives

Guidelines for developing water quality objectives have been suggested by a number of sources; the most comprehensive is likely that proposed by the U.S. EPA (U.S. EPA, 1979a). However, even this is not the definitive text on the topic, for a number of major problems still remain unresolved and/or subject to judgement calls by the guideline formulator. Problems specific to the chlorinated benzenes study are outlined below.

The major problems faced in the formulation of guidelines for chlorinated benzenes stems from the fact that only an incomplete data set exists - i.e. data on an all compounds and isomers are not available - and from the fact that a variety of durations and responses were employed in obtaining the experimental data that do exist. A complicating factor is that there appears to be significant differences between measured and nominal (i.e. calculated but unmeasured) exposure concentrations, the latter being higher. This makes the accuracy of tests employing nominal concentrations questionable, especially for the more hydrophobic compounds.

In addition to these problems are the more usual problems encountered in the interpretation of toxicity data: differences in species responses, differences in sensitivity with age and condition, influence of water quality, purity of the test chemical preparations, and others.

In the simplest, traditional approach water quality objectives could be set simply by applying a safety factor to the lowest concentration eliciting a chronic response or by applying an application factor and a safety factor to the lowest concentration eliciting an acute response. This would be done for those compounds and isomers where data are available, where data are insufficient no guidelines would be set. However, a relatively new approach to aquatic toxicity interpretation (although well established in human pharmacology), Quantitative Structure-Activity Relationships (QSAR) has recently been the focus of a great deal of scientific research and discussion (Veith et al., 1983; Konemann, 1980; Spacie and Hamelink, 1982; among others) and is proposed for use in this case.

The QSAR approach is thought to generate acute and chronic toxicity estimates that fall within the range that would be expected to be obtained from typically employed toxicity test procedures. In fact, the QSAR approach is based on an observed relationship between K_{ow}, the octanol/water partition coefficient, a measure of the hydrophobicity of the chemical and actual toxicity test results. For acute toxicity the model used is that proposed by Veith et al. (1983). For chronic toxicity the model proposed by McCarty et al. (unpublished) is employed. Other researchers (Call el al., 1983) working independently have reported similar, although less extensive, QSARs for chronic toxicity. Since these QSAR models were developed from actual experimental observations they are subject to and include errors from a variety of sources as discussed earlier. Therefore error about the line defining the relationship is relatively large yet the general trends which will be presented in Figure 1 are still apparent.

The chlorinated benzenes are an ideal group to apply QSAR methodology to, in that they are, as previously discussed, a closely related group in chemical, physical, and toxicological terms. In fact, data on several chlorinated benzenes were used by Veith et al. (1979a, 1983) in developing the initial QSAR for acute toxicity. The whole chlorinated benzene group appears to fall within the characteristics used by Veith et al. (1983) to define the type of chemicals to which this QSAR might be applicable. The group is defined as organic chemicals which produce toxic effects by narcosis. Narcosis is defined as a reversible, chemically non-specific, physiological effect, independent of chemical structure as long as specific pharmacophores (certain chemical groups which are known to substantially increase toxicity) are not present.

It must be stressed that the QSAR used for objective development herein is not to be considered a complete substitute for experimental data. Rather at this time it must be viewed as a means of estimating values and rationalizing incomplete data sets to enable objectives to be developed. As more data are generated the guidelines developed by means of this methodology must be reviewed and updated.

4.2.1 Development of Objectives Based on Fish Toxicity

As discussed in Section 2.3 the toxicity data base for fish is judged to be the most complete and most accurate. Data for organisms other than fish is not included in an attempt to reduce variability. However, an inspection of this data will verify that the general trends apparent in the fish data

are also apparent for other types of organisms. Also there are no data to suggest that any other organisms other than fish are more sensitive to the effects of chlorinated benzenes.

The data used in the development of objectives has been rigorously screened to ensure that it is compatible. The bases for this screening was as follows:

- only data based on measured concentrations was included, no data from tests where the toxicant concentration was not measured in the test was used.

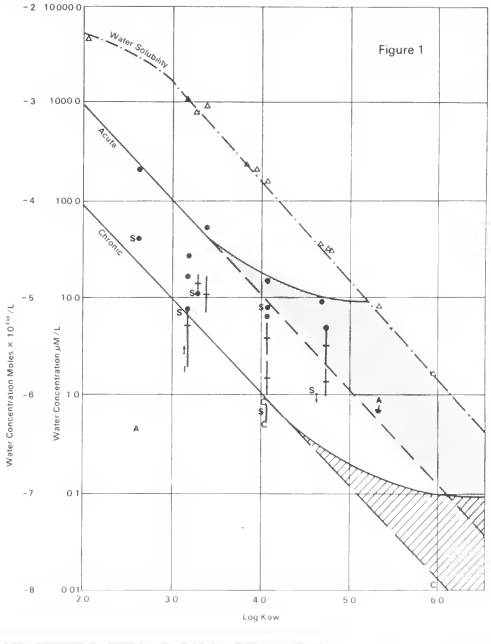
- only 96 h LC_{50} tests were included in the acute toxicity data used.

- only results from relatively long-term early life stage tests were included in the chronic toxicity data, although not all tests employed the same protocol or exposure duration.

The data from Tables 11 and 13 which pass the selection process are presented again in Table 16. In Table 16 the data have been transformed from a weight per volume basis (g/L) to molarity (moles/L) as it is well established in pharmacology that toxicity is related to the number of molecules present not the weight. Also presented in Table 16 are the lowest concentrations reported to cause an effect if not already included in the table. This is to include the results of non-standard tests which would not otherwise pass the selection process but still may have some use in the interpretative process. All of this data is plotted on Figure 1 as are estimated water solubility values for each compound.

Legend

△	Calculated, corrected water solubility (See Table 2, note 3)
●	96 h LC_{50} estimates of acute toxicity (See Table 16)
†	Chronic toxicity estimates (See Table 16)
A	Lowest reported concentration causing acute response in fish, if from non-standard test
C	Lowest reported concentration causing chronic response in fish, if from non standard test
S	Indicates test was on a salmonid fish
Acute	Solid line indicates relationship between acute toxicity (96 h estimates) test results and K_{ow} demonstrated by Veith et al. (1983) for 65 industrial chemicals
Chronic	Solid line indicates hypothetical relationship between standard chronic toxicity test results and K_{ow} proposed by McCarty et al. (manuscript)
Chronic / Acute	Shaded areas represent the range of K_{ow} values in which the net bioaccumulation rate may limit the expression of toxicity due to the relatively short duration of standard tests, for acute and chronic toxicity, respectively
	The broken straight lines extending from the point where curvature starts, for both the acute and chronic toxicity relationships, indicates the relationship between toxicity and K_{ow} that might be expected, for both cases, where time is not limiting bioaccumulation and the ultimate level of bioaccumulation is achieved in the exposed organism



Water Solubility and Acute and Chronic Toxicity Information for Chlorinated Benzenes

In plotting Figure 1 the K_{ow} values used are those calculated by the method of McKay (1982) based on his corrected water solubility estimates (see Table 2). The advantage of this approach is a more realistic estimate of K_{ow} which is isomer specific. Typically employed methods (Leo et al., 1971) give identical K_{ow} values for isomers of a compound (see Table 2), yet solubility and toxicity data suggest there should be some differences.

In addition to the data mentioned above being plotted on Figure 1, the QSAR relationships are also indicated. The acute toxicity relationship plotted is that proposed by Veith et al. (1983). The chronic toxicity relationship is that proposed by McCarty et al. (unpublished). Inspection of Figure 1 suggests that the acute and chronic data for chlorinated benzenes follow the trends indicated by the respective QSAR lines proposed for selected groups of organic chemicals.

The shaded portions of Figure 1 represent areas where the results of acute or chronic tests are thought to become limited by the standard test exposure times. Consequently, in this area the toxicity test is thought to be underestimating what the toxicity would be in an exposure not limited by time. Under real world conditions the toxicity relationship would not be expected to curve towards the solubility limit of the chemical (known as the Ferguson Solubility Cut-off) but rather be a continuation of the straight line relationship, as indicated.

Therefore, in the shaded areas of the graph an acute or chronic toxicity estimate thought to be more representative of what might be expected in a real freshwater environment,

and subsequently more valid for objectives formulation, should be that indicated by the straight extensions of the QSAR lines (McCarty et al., unpublished).

The short time frame of acute testing, which has often been used in guideline formulation, can introduce significant error as uptake rates and partitioning, among other things, are not taken into account (Veith and Konasewich, 1975). For the chlorinated benzenes, and in fact many toxic organic compounds, the information derived from longer term chronic toxicity tests is more useful than acute tests because it better reflects the complex nature of the interactions of these compounds over a longer time frame and is more readily applicable to field situations. Chronic toxicity estimates are therefore a more logical choice as a basis for estimating "safe" environmental levels, incorporating, of course, the caution discussed in the previous paragraph.

Table 17 contains chronic toxicity estimates based on interpolation from Figure 1 using the K_{ow} values given in Table 16. To obtain the proposed safe water concentration a safety factor of 0.10 is applied to each chronic estimate for each individual compound. By this method all proposed safe values are below the lowest reported value causing an effect thus leaving some "buffer" to allow for more sensitive species than those tested; that is, with the exception of monochlorobenzene.

As discussed in Section 2.3 some data were generated by Black et al. (1982) and Birge et al. (1979), using a special apparatus which limited volatility and likely increased the bioavailability of the monochlorobenzene to the test

TABLE 17

ESTIMATED SAFE LEVELS FOR CHLORINATED BENZENES
BASED ON AQUATIC TOXICITY INFORMATION

COMPOUND	ISOMER	MOLECULAR WEIGHT g/MOLE	CHRONIC TOXICITY[1] ESTIMATE μ MOLES/L	SAFETY FACTOR	ESTIMATED SAFE LEVEL μ MOLES/L	μg/L
Monochlorobenzene	-	112.56	25	0.10	2.5	281
Dichlorobenzene						
	1,2	147.01	5.0	0.10	0.50	74
	1,3	147.01	4.3	0.10	0.43	63
	1,4	147.01	7.0	0.10	0.70	103
Trichlorobenzene	1,2,3	181.45	1.7	0.10	0.17	31
	1,2,4	181.45	0.95	0.10	0.095	17
	1,3,5,	181.45	1.2	0.10	0.12	22
Tetrachlorobenzene	1,2,3,4	215.9	0.20	0.10	0.020	4.3
	1,2,3,5	215.9	0.19	0.10	0.019	4.1
	1,2,4,5	215.9	0.25	0.10	0.025	5.4
Pentachlorobenzene	-	250.34	0.060	0.10	0.006	1.5
Hexachlorobenzene	-	284.79	0.015	0.10	0.0015	0.43

NOTE: 1. Values interpolated from chronic toxicity line on Figure 1. Straight portion of line below K_{ow} of about 4.5 used rather than curved portion.

organisms. It appears that this data is not readily comparable to that obtained by standard methodologies. Given the volatile nature of monochlorobenzene, its very short half life in water, its low K_{ow} and K_{oc}, which prevent it from accumulating in the aquatic environment, it is likely safe to continue with the number derived by the QSAR approach.

The advantage of using the QSAR methodology to estimate chronic toxicity, and from this estimate a "safe" level can be seen when the traditional methodology, based on acute toxicity (Ontario Ministry of the Environment, 1978), is reviewed. The methodology is:

> "Concentrations of materials that are non-persistent or have non-cumulative effects should not exceed 0.1 of the 95 h LC_{50} at any time or place after mixing with the receiving water. The 24 h average of the concentration of these materials should not exceed 0.05 of the 96 h LC_{50} after mixing. For toxicants which are persistent or cumulative, the concentrations should not exceed 0.05 of the 96 h LC_{50} at any time or place nor should the 24 h average concentration exceed 0.01 of the 96 h LC_{50}".

There may be some difficulty in assigning certain of the chlorinated benzenes to the persistent or non-persistent category, as the definition is not standardized (Munro et al., 1983. Ralston, personal communication). Similarly, the definition of what degree and test methodology best categorizes bioaccumulative and non-bioaccumulative is not standardized. For this reason, the most stringent case - i.e. 0.01 of the 96 h LC_{50} - will be applied to all.

Using an application factor of 0.01 and the 96 h LC_{50} value for any chemical, where both actual acute and chronic toxicity data are available (see Figure 1), will generate an estimated water quality objective which is within the range known to produce chronic effects. For the less stringent case, 0.1 of the 96 h LC_{50}, the values are obviously also within the known chronic toxicity range. Clearly, the traditional method of generating water quality objectives from acute toxicity data is not applicable to the chlorinated benzenes and in this case cautions presented in Ontario Ministry of the Environment (1978) concerning this approach are particularly pertinent.

4.2.2 Other Considerations

It is felt that the safe levels suggested in Table 17 would be sufficient as objectives for the protection of all aquatic organisms as there is some buffer to allow for more sensitive organisms. However, another consideration in setting water quality objectives must be made; that is, food chain bioaccumulation and the resultant effects on higher consumers.

Hexachlorobenzene is particularly problematic in this area. Several guidelines for human consumption of foodstuffs have been formulated. These guidelines are reported below:

300 µg/Kg for HCB in meat fat - Louisiana State Department of Agriculture

300 µg/Kg for HCB in cows milk - Australia NHMRL

20 µg/Kg for HCB in whole cows milk - WHO

100 µg/Kg for HCB in eggs - Australia NSWDH

500 µg/Kg for HCB in milk and egg fat - FAO/WHO

1000 µg/Kg for HCB in meat and poultry fat - FAO/WHO

All of these levels are cited in U.S. EPA (1980a).

Although the exact details of the development of these guidelines for human consumption are not readily available, the general approach used was likely similar to that which follows. The research data on which the guideline is based is probably from laboratory animals. For HCB chronic effects on liver function and reproduction have been reported and in addition it is considered to be a carcinogen (U.S. EPA, 1980a). The extrapolation of this lab data to humans, with appropriate considerations and safety factors would generate an acceptable daily intake. For example FAO/WHO (1974) proposed an acceptable daily intake (ADI) of 2.4 µg/Kg/day for HCB. The level in foodstuffs could then be set by estimating the typical daily consumption of the appropriate foodstuffs by the average (70 kg) man, and allotting portions of the ADI. Where children are major consumers, in the case of milk for example, levels are often set lower than for adults to allow for their smaller body weight (U.S. EPA, 1980a).

As discussed in Section 2.4, there is some indication that a level of about 1000 µg/Kg of HCB in the diet may be a rough estimate of a safe level of consumption for birds. This value is in reasonable agreement with the values suggested above for human foodstuffs.

It is possible to calculate, by using the K_B (bioconcentration factor) a water concentration of hexachlorobenzene which will produce an average body burden of no higher than, for example 300 µg/Kg, which is roughly the mean of the six human consumption guidelines mentioned above. The formula is

$$\text{water concentration } (\mu g/L) \approx \frac{\text{body concentration } (300 \mu g/Kg)}{K_B}$$

If we use a log K_B of 4.66 from Table 9, the water concentration which is derived for HCB is 0.0066 µg/L, approximately 66 times less than the "safe" value derived from the toxicity information.

The chlorinated benzenes appear to be a closely related group of chemicals, and as discussed in Section 2.3 appear to act in a similar manner, in a toxicological sense. For this reason the chlorinated benzenes other than HCB were examined for biaccumulation hazard by means of the above formula. A similar body burden level, 300 µg/Kg, was employed along with the appropriate K_B values from Table 9. The following results were obtained:

- QCB - 0.031 µg/L, about 50 times less than the toxicity-derived safe value
- 1,2,3,4 TeCB - 0.12 µg/L, about 37 times less than the toxicity-derived value
- 1,2,3,5 TeCB - 0.11 µg/L, about 37 times less than the toxicity-derived value
- 1,2,4,5 TeCB - 0.13 µg/L, about 40 times less than the toxicity-derived value
- 1,2,3 TCB - 0.89 µg/L, about 35 times less than the toxicity-derived value
- 1,2,4 TCB - 0.57 µg/L, about 30 times less than the toxicity-derived value

- 1,3,5 TCB - 0.72 µg/L, about 31 times less than the toxicity-derived value

- 1,2 DCB - 3.2 µg/L, about 23 times less than the toxicity-derived value

- 1,3 DCB 2.9 µg/L, about 22 times less than the toxicity-derived value

- 1,4 DCB 3.8 µg/L, about 27 times less than the toxicity-derived value

- MCB - 15 µg/L, about 18 times less than the toxicity-derived safe value

Although as noted above there is only evidence sufficient to allow bioaccumulation hazard assessment for hexachlorobenzene, other information presented in this report suggests that a similar hazard, the degree of which is a function of bioaccumulation potential, may exist for other chlorinated benzenes.

To allow for possible bioaccumulation hazards it is recommended in the final criteria development that the estimated "safe" concentrations proposed in Table 17, which already include a safety factor of 0.10, incorporate additional safety factors: 0.055 for monochlorobenzene, 0.037 for dichlorobenzenes, 0.029 for trichlorobenzenes, 0.25 for tetrachlorobenzenes, 0.020 for pentachlorobenzene and 0.015 for hexachlorobenzene. The additional safety factor values are approximately the inverse of the amount, by which the bioaccumulation hazard derived values exceed the toxicity-

derived values. For the compounds where there are isomers - DCB, TCB, and TeCB - the most conservative of the values obtained earlier in the calculations was applied to all isomers of the compound.

The above methodology is suggested with the acknowledgement that no standardized approach for dealing with bioaccumulative materials has been proposed. However, a precedent has been established in the formulation of water quality objectives for mercury and PCBO. In these cases, food chain bioaccumulation effects on higher trophic level consumers - birds and mammals including humans - were taken into account in guideline formulation (MOE, 1979).

It should be noted that this simple procedure does not take into account a variety of factors, such as differing proportions of body fact, differing physical sizes, differing diets, among other things, and assumes that all toxicant input is from water alone. These and other factors do affect the rate of net bioaccumulation and the ultimate bioaccumulation level and subsequently toxicity but the state of the art of understanding of their influences is poor and other than this caveat, further sophistication is unwarranted at this time.

4.2.3 Suggested Provincial Water Quality Objectives for Chlorinated Benzenes

The suggested objectives are presented in Table 18. These values were generated by taking the safe level estimates presented in Table 17, adjusting these values by a constant safety factor (0.1) and then further adjusting these values by an additional safety factor, as discussed in 4.2.2, to

TABLE 18

SUGGESTED PROVINCIAL WATER QUALITY OBJECTIVES
FOR CHLORINATED BENZENES

COMPOUND	ISOMER	SUGGESTED CRITERIA µg/L
Monochlorobenzene	-	15
Dichlorobenzene	1,2	2.5
	1,3	2.5
	1,4	4.0
Trichlorobenzene	1,2,3	0.90
	1,2,4	0.50
	1,3,5	0.65
Tetrachlorobenzene	1,2,3,4	0.10
	1,2,3,5	0.10
	1,2,4,5	0.15
Pentachlorobenzene		0.030
Hexachlorobenzene		0.0065

account for the hazards presented by bioaccumulation. The final total safety factors (which are products of the two component factors) applied to the chronic toxicity estimates of Table 17 are therefore:

	Aquatic Toxicity Safety Factor		Bioaccumulation Hazard Safety Factor		Total Safety Factor
MCB	0.10	x	0.055	=	0.0055
DCBs	0.10	x	0.037	=	0.0037
TCBs	0.10	x	0.029	=	0.0029
TeCBs	0.10	x	0.025	=	0.0025
QCB	0.10	x	0.020	=	0.0020
HCB	0.10	x	0.015	=	0.0015

The values obtained by multiplying the overall safety factor by the chronic toxicity estimates (for each component and isomer) from Table 17 are then converted from moles/L to µg/L, rounded to the nearest value with two significant digits ending in a 0 or 5, as appropriate, to generate the proposed criteria which appear in Table 18.

4.3 Criteria Developed by Other Jurisdictions

A summary of guideline criteria or objectives for various chlorinated benzenes in water appears in Table 19. These guidelines were formulated for the protection of human health from the adverse effects of ingesting chlorinated benzenes in water and aquatic organisms.

HCB is considered to be a potential carcinogen, although sufficient information to definitely confirm this is not yet available (U.S. EPA, 1980a). For this reason the guideline criterion for this compound is presented for various levels of risk as well as the not detectable guideline mentioned above. For example a 10^{-5} risk level means that at the water concentration given, an increase in the cancer rate equivalent to one additional cancer death per 100 000 people would be expected.

With only one exception, no guidelines currently exist for the protection of aquatic life from the adverse effects of chlorinated benzenes. A tentative water quality criteria of 50 µg/L was proposed for the protection of aquatic life from the adverse effects of 1,4-DCB. This was based on research carried out for the Italian National Research Council (Calamari et al., 1982).

Initially, in 1979 the U.S. EPA (1979a) proposed criteria for the protection of freshwater aquatic life for MCB, DCB, TCB, and HCB. The 24-hour average exposure levels were set at 1500 µg/L, 310 µg/L, 210 µg/L and not detectable (at a detection limit of 1 µg/L), respectively. However, these criteria were omitted from the 1980 versions of the Ambient Water Quality Criteria documents (U.S. EPA, 1980a,b) as upon review it was judged that the data base was not sufficient to support the formulation of these guidelines.

section 5

AREAS FOR SUGGESTED RESEARCH

7.0 AREAS FOR SUGGESTED RESEARCH

1. To improve the accuracy of the objectivies and to further validate the QSAR approach used in the objective development, one or more complete data sets - i.e. all 12 compounds plus the parent compound, benzene - are needed for a single fish species. A salmonid species would be a good first choice since they appear to be a sensitive species. The test employed should be of the long-term chronic variety, either a complete life cycle or an ELS test plus adult tests.

   In addition to the above tests toxicity information on other aquatic organisms, including, algae, macrophytes, invertebrates and bacteria, should be collected employing a similar methodology to ensure that the results are comparable.

2. As a part of toxicity testing histological and biochemical informations should be collected simultaneously, as should data on bioaccumulation and distribution within the body of the organism. The whole body, organ, and/or tissue concentrations at which typically determined effects on viability, growth reproduction, etc. become evident should be determined as a part of the toxicity test. In addition to total concentrations, the amount of lipid must be reported for each determination so that data may be corrected to a common lipid level or expressed on a per unit lipid basis.

The mode or modes of toxicity by which chlorinated benzenes exert their adverse effects could be better defined by this type of information. As well, this data could be used to further investigate the apparent simple additivity of the toxic effects of different chlorinated benzenes. Such a clarification would be a major improvement in the understanding of the joint toxicity of multiple compounds.

3. Since the rate and ultimate level of bioaccumulation are both important factors in determining the toxic effects on biota, future toxicity studies should quantify net bioaccumulation levels at various exposure times to determine if equilibrium has been reached and/or to allow the relative exposure to be calculated. The determination of uptake and depuration rate constants under a variety of test conditions is an alternative means of achieving this goal.

   Such data would reduce the difficulties in interpreting toxicity test data caused by the standard practice of using constant exposure times. This procedure is not valid when the toxic materials accumulate at different rates, and therefore the test organisms have not been exposed to an equivalent amount of toxicant.

4. Further study on the partitioning of hydrophobic organic chemicals between the water and the non-living organic (hydrophobic) phase in the water should be carried out. This partitioning appears to have a major influence on bioavailability, and hence on uptake and subsequently on the expression of toxic effects (see Section 2.3). Therefore, the bioavailability of organic toxicants

associated with dissolved (humic and fulvic acids for example) and particulate organics in the water and with that simply dissolved in the water should be estimated in toxicity testing. Information on the nature and amount of organic material in water should be collected from various areas in Ontario to enable the laboratory data to be extrapolated to the field situation when the experimentation has been carried out.

5. The aquatic fate of chlorinated benzenes must be more accurately determined. It is important to know where a compound introduced into the aquatic environment goes; whether it evaporates to the atmosphere, partitions to sediments, biota or other organics, or remains dissolved in the water column. The time course of these events and the persistence relative to physical and biological degradation mechanisms, is also important.

Realistic models based on known chemical and physical characteristics, refined by means of field observations, would be a major tool that would likely be applicable to a variety of organic compounds. When refined, such models on fate combined with models on bioaccumulation and toxic effects would be a very important interpretive and predictive tool. These models could greatly enhance the ability to develop adequate Water Quality Objectives for the increasing larger number of organic chemicals found in the aquatic environment.

section 6

APPENDIX - TABLES

TABLE 1

PRODUCTION AND USES OF CHLORINATED BENZENES

COMPOUND	ISOMER	PRODUCTION	REFERENCE	BY PRODUCTION	REFERENCE	USES	REFERENCE
Monochlorobenzene	MCB	• worldwide production - 600,000 t per year	WHO, 1980	•contaminant of DDT	Ware and West, 1977	•aynthetic intermediate in production of phenol, DDT, nitrochlorobenzene	Ware and West, 1977
		• USA production - 300 million pounds per year				•solvent in manufacture of adhesives, paints, polishes, waxes, diisocyanates, pharmaceuticals and natural rubber	Ware and West, 1977
		• no Canadian production	MOE, 1978			•flotation medium •perfume intermediate	WHO, 1980
Dichlorobenzene	1,2-DCB	• USA production (1975) - 54-55 million pounds • no Canadian production	Ware and West, 1977 MOE, 1978	•almost entirely as a by-product of monochloro-benzene production	U.S E.P.A. 1980(a)	•major use as a solvent in toluene diisocyanate production •solvent in textile industry •degreaser for leather and metals (in increasingly less frequent use in automobile and aircraft engines) •intermediate in herbicides, dyes and epoxy tar production •solvent in toilet bowl cleaner •minor uses as odourant, household pesticide and intermediate in rust proofing	Ware and West, 1977

TABLE 1 (Page 2)

PRODUCTION AND USES OF CHLORINATED BENZENES

COMPOUND	ISOMER	PRODUCTION	REFERENCE	BY PRODUCTION	REFERENCE	USES	REFERENCE
Dichlorobenzene	1,3-DCB	• no Canadian production	MOE, 1978	•may occur as a contaminant of 1,2 or 1,4 DCB formulations	U.S. EPA, 1980(b)	•information not available	U.S. EPA, 1980(b)
Dichlorubenzene	1,4-DCB	• USA production - 40 million pounds • no Canadian production	Ware and West, 1977	•almost entirely produced as a byproduct of monochlorobenzene production	U.S. EPA, 1980(b)	•major uses as deodorant and moth repellent •fumigant to control lice and cockroaches •used in the process of molding grinding wheels and molding concrete and stoneware •fungistat and insecticide •minor uses as an extreme pressure lubricant and as an intermediate in polyphenylenesulfide and 4-chloro-2 nitroaniline production	Ware and West, 1977
Trichlorobenzene	1,2,3-TCB	-	-			•minor component of a solvent •used also as a lubricant and dielectric fluid •minor importance as intermediates in chemical syntheals	Ware and West, 1977

TABLE 1 (Page 3)

PRODUCTION AND USES OF CHLORINATED BENZENES

COMPOUND	ISOMER	PRODUCTION	REFERENCE	BY PRODUCTION	REFERENCE	USES	REFERENCE
Trichlorobenzene	1,2,4-TCB	• USA 1973 production – 28 million pounds	Ware and West, 1977	–	–	•major use as a dye carrier in the application of dyes to polyester materials •intermediate in synthesis of herbicide •solvent for the functional crystallization of high melting compounds •potential use as dielectric fluids for transformers flame retardant, degreaser solvent, heat transfer medium •insecticide	Ware and West, 1977 WHO, 1980
Trichlorobenzene	1,3,5-TCB	–	–			•minor use as intermediate in chemical synthesis	Ware and West, 1977
Tetrachlorobenzene	TeCB	–	–	•Small quantities from photodecomposition of pentachloronitrobenzene	Ware and West, 1977	–	–
Tetrachlorobenzene	1,2,3,4-TeCB	–	–	–	–	–	

TABLE 1 (Page 4)

PRODUCTION AND USES OF CHLORINATED BENZENES

COMPOUND	ISOMER	PRODUCTION	REFERENCE	BY PRODUCTION	REFERENCE	USES	REFERENCE
Tetrachlorobenzene	1,2,4,5-TeCB	• USA production (1973) ~18 million pounds	Lewis, 1975	-	-	•intermediate in production of 2,4,5-trichlorophenol (TCP) which is used as a fungicide, disinfectant, preservative for leather wool, and textiles and is an intermediate in production of herbicides and insecticides •fire retardant used to impregnate materials for moisture resistance •dielectric fluid	Ware and West, 1977
Pentachlorobenzene	QCB	• USA - 1972 production < 1 ton	Mumma and Lawless, 1975	•by-product of HCB, up to 10% contamination •small quantities from photodecomposition of pentachloronitrobenzene or as a contaminant •may be formed from decomposition of hexachlorobenzene in the environment	Ware and West,	•intermediate in production of fungicide pentachloronitrobenzene	Ware and West, 1977

TABLE 1 (Page 5)

PRODUCTION AND USES OF CHLORINATED BENZENES

COMPOUND	ISOMER	PRODUCTION	REFERENCE	BY PRODUCTION	REFERENCE	USES	REFERENCE
Hexachlorobenzene	HCB	• USA production 1973 - 350 ton	Mumma and Lawless, 1975	•almost entirely as waste or a byproduct of chlorinated hydro - carbon processes production of perchloro-ethylene, carbon tetra-chloride, tri-chloroethylene	Mumma and Lawless, 1975	•principal use as a fungi-cide to control wheat but and smut fungi of other grains •additive for pyrotechnic compositions for the military •porosity controller in manufacturer of electrocodes •chemical intermediate in dye manufacturer and organic synthesis •peptizing agent in production of tires •wood preservative •intermediate for penta-chlorophenol production	Mumma and Lawless, 1975
			Mumma and Lawless, 1975	•byproduct of pentachloro-benzene and chloride gas •contaminant of the pesticides atrazine, propazine, simazine, dacthal, mirex and pentachloro-nitrobenzene			
				•byproduct of incineration of domestic and industrial wastes	EPA, 1976		
Hexachlorobenzene	HCB	• Not produced directly as a product in Canada	Gilbertson, 1978	•3900 metric tonnes in-directly pro-duced annually in the USA as waste by-product or impurity	Nimi and Cho, 1981		

TABLE 2

CHEMICAL AND PHYSICAL PROPERTIES OF CHLORINATED BENZENES

COMPOUND/SYNONYMS	ISOMER	MOLECULAR WEIGHT	DENSITY (g/ml) @20°C	MELTING POINT (C°)	BOILING POINT (C°) @760 mm Hg	VAPOUR PRESSURE (mm Hg) @20-25°C	HENRY'S LAW CONSTANT kPa m^3/mol [1]
Monochlorobenzene	MCB	112.56	1.1058	-45.6	132	9	0.363
Phenylchloride Chlorobenzene Chlorobenzol Benzene monochloride							
Dichlorobenzene							
ortho-Dichlorobenzene	1,2-DCB	147.01	1.3048	-17.0	180.5	1.28	0.195
meta-Dichlorobenzene	1,3-DCB	147.01	1.2884	-24.7	173	1.89	0.366
para-Dichlorobenzene	1,4-DCB	147.01	1.2475	53.1	174	0.4	0.160
Trichlorobenzene	1,2,3-TCB	181.45		53-4	218-9	0.07	0.234
	1,2,4-TCB	181.45	1.4542	16.95	213.5	0.29	0.379
	1,3,5-TCB	181.45		63	208	0.15	0.161
Tetrachlorobenzene	1,2,3,4-TeCB	215.9		47.5	254		0.261
	1,2,3,5-TeCB	215.9		54.5	246		0.593
	1,2,4,5-TeCB	215.9	1.858(22°C)	140	243	0.05	0.261
Pentachlorobenzene	QCB	250.34	1.8342(16.5°C)	86	277		0.977
Hexachlorobenzene	HCB	284.79	1.5691	230	322	1.09×10^{-5}	0.005
Perchlorobenzene			(23.6°C)		(sublimes)		

Source: Weast, 1977: Jacobs, 1957: U.S. EPA, 1980 a,b: Oliver and Niimi, 1983: U.S. EPA, 1977.

1. Mackay and Shiu, 1981; means of calculated values

..... 2

TABLE 2 (Page 2)

CHEMICAL AND PHYSICAL PROPERTIES OF CHLORINATED BENZENES

	WATER SOLUBILITY @ 25°C (µg/L)				LOG K_{ow}[1]		
COMPOUND/ISOMER	MEASURED	(REPORTED RANGE)	MEASURED[2]	CORRECTED[3] mol/m³	MEASURED[4]	CALCULATED[5]	CALCULATED[6]
Monochlorobenzene							
MCB	488,000[7]	100,000[8] 500,000[9]	491,000	4.36	2.83	2.84	2.61
Dichlorobenzene							
1,2-DCB	145,000[9]		134,000	0.912	3.40	3.38	3.29
1,3-DCB	123,000[9]		122,000	0.830	3.44	3.38	3.34
1,4-DCB	70,000[10]	60,000[10] 87,000[11]	83,100	1.07	3.37	3.39	3.22
Trichlorobenzene							
1,2,3-TCB	-		24,100	0.252	4.11	4.26	3.85
1,2,4-TCB	25,000[12]	25,000[13] 30,000[14]	29,800	0.164	4.02	4.26	4.04
1,3,5-TCB	-	-	15,800	0.207	4.15	4.26	3.94
Tetrachlorobenzene							
1,2,3,4-TeCB	-	-	4,310	0.0333	4.46	4.97	4.73
1,2,3,5-TeCB	-	-	3,500	0.0317	-	4.97	4.75
1,2,4,5-TeCB	400-500[15]		595	0.0379	4.52	4.97	4.67
Pentachlorobenzene							
QCB	-	-	560	0.00899	4.94	5.68	5.31
Hexachlorobenzene							
HCB	6[16]	6-35[17]	5	0.00188	5.50	6.39	5.98

NOTES:

1 Logarithm of the octanol/water partition coefficient.
2 Mean of values reported by Mackay and Shiu, 1981.
3 Compounds liquid at 25°C not corrected, solids are corrected to estimated subcooled solubility by method of Mackay, 1982.
4 Values compiled by Oliver and Niimi, 1983 as "best" of available measured data, except MCB which is from USEPA 1980a.
5 From Neely et al., 1974 and Smith, 1981, based on the method of Leo et al., 1971 and Hansch et al., 1972.
6 K_{ow} calculated from mean of values reported by Mackay and Shiu, 1981 by method of Mackay 1982.
7 Marsden and Marr, 1963.
8 Lu and Metcalf, 1975.
9 Verschueren, 1977.
10 Aquatic Toxicity Research Group, 1982a.
11 Aquan-Yuen et al., 1979.
12 Branson, 1977.
13 Aquatic Toxicity Research Group, 1982b.
14 Dow, 1978.
15 Aquatic Toxicity Research Group, 1982c.
16 Laseter et al., 1976.
17 Neely, 1980.

TABLE 3

SUMMARY OF OCCURRENCE OF CHLORINATED BENZENES IN WATER

Compound	Isomer	Raw Water Great Lakes	Raw Water Canada other than Great Lakes	Raw Water USA	Ground Water USA	Sewage effluent/ Municipal Waste Canada	Sewage effluent/ Municipal Waste USA	Industrial discharge/ waste Canada	Industrial discharge/ waste USA	Drinking Water Canada	Drinking Water USA
Monochlorobenzene	MCB			4.7 µg/L	1.0 µg/L		0.1-27.0 µg/L		0.1-17.0 µg/L	ND-0.021 µg/L	
Dichlorobenzene	DCB				1.2-12 µg/L	ND-32 µg/L (raw) ND-10 µg/L (final effluent) (DL=0.05 µg/L)		ND-138 µg/L DL= 0.05 µg/L	0.9-33 µg/L	ND-0.01 µg/L	
	1,2-DCB	ND-0.007 µg/L (DL= 0.001 µg/L)	ND-0.031 µg/L (DL= 0.001 µg/L		1-71 µg/L	0.006-0.022 µg/L	<0.11-12 µg/L		15-690 µg/L	ND-0.007 (DL=0.001 µg/L	1-2.5 µg/L
	1-3-DCB	ND (DL=0.001 µg/L)	ND-0.004 µg/L (DL= 0.001 µg/L		0.5 µg/L	0.007-0.014 µg/L				ND-0.002 µg/L (DL=0.001 µg/L	0.10- <3 µg/L
	1-4-DCB	0.003-0.064 µg/L	ND-0.042 µg/L (DL= 0.001 µg/L		0.5 µg/L	0.048-0.920 µg/L	0.42-12 µg/L		58 µg/L	0.008-0.020 µg/L	0.07-1 µg/L
Trichlorobenzenes	1,2,3-TCB	ND-0.0002 µg/L (DL= 0.0001 µg/L)	ND-0.0007 µg/L (DL= 0.0001 µg/L)			0.002-0.003 µg/L	21-46 µg/L			0.0001-0.001 µg/L	
	1,2,4-TCB	0.0001-0.001 µg/L	ND-0.008 µg/L (DL= 0.0001 µg/L)			0.005-0.018 µg/L	<0.01-6.0 µg/L		12-500 µg/L	0.001-0.004 µg/L	
	1,3,5-TCB	ND-0.0004 µg/L (DL= 0.0001 µg/L)	ND (DL= 0.0001)			0.0001-0.0005 µg/L	<0.01-0.8 µg/L			ND (DL= 0.0001 µg/L	
	TCB							ND-80 µg/L			
Tetrachlorobenzene	1,2,3,4-TeCB	ND-0.0005 µg/L (DL= 0.00005 µg/L	ND-0.0002 µg/L (DL= 0.00005 µg/L			0.0002-0.003 µg/L				0.0001-0.0004 µg/L	
	1,2,3,5-TeCB	ND (DL= 0.00005 µg/L)	ND (DL= 0.00005 µg/L			0.0001-0.001 µg/L				ND (DL= 0.00005 µg/L	
	1,2,4,5-TeCB	ND-0.0003 µg/L (DL= 0.00005 µg/L)	ND (DL= 0.00005 µg/L			0.0003-0.001 µg/L				ND-0.0003 µg/L (DL= 0.00005 µg/L)	
Pentachlorobenzene	QCB	ND-0.0006 µg/L (DL= 0.00001 µg/L)	ND-0.0001 µg/L (DL= 0.00001 µg/L)			0.0005-0.001 µg/L	detected			0.00003-0.00005 µg/L	
Hexachlorobenzene	HCB	0.00001-0.001 µg/L	0.00002-0.0001 µg/L	0.1-72.8 µg/L (in industrialized area)		0.001-0.002 µg/L		0.005-74.9µg/L		ND-0.005 µg/L	0.006-0.01 µg/L

ND - not detected
DL - detection limit

TABLE 3 (Page 2)

DETAILS OF OCCURRENCE OF CHLORINATED BENZENES IN WATER: NORTH AMERICA

COMPOUND	ISOMER	LOCATION	SOURCE	CONCENTRATION	REFERENCE
Monochloro-benzene	MCB	Miami, FL	Ground water	1.0 µg/L	U.S. EPA, 1975; 1977
	MCB	Philadelphia, PA	Raw water contaminated with municipal waste	0.1 µg/L	U.S. EPA, 1975; 1977
	MCB	Cincinnati, OH	Raw water contaminated with industrial discharge	0.1-0.5 µg/L	U.S. EPA, 1975; 1977
	MCB	New York, NY	"Uncontaminated" upland water	4.7 µg/L	U.S. EPA, 1975; 1977
	MCB	Lawrence, MA	Raw water contaminated with industrial discharge	0.12 µg/L	U.S. EPA, 1975; 1977
	MCB	Terrebone Parish, LA	Raw water contaminated with municipal waste	5.6 µg/L	U.S. EPA, 1975; 1977
	MCB	Lawsons Fork Creek, SC	Industrial discharge	8.0-17.0 µg/L	U.S. EPA, 1975; 1977
	MCB	Coosa River, GA	Municipal discharge	27.0 µg/L	U.S. EPA, 1975; 1977
Dichloro-benzene	DCB	Michigan	Chemical plant wastewater (seepage and cooling)	10 µg/L	U.S. EPA 1977
	DCB	Kentucky	Ground water	1.2, 12 µg/L	U.S. EPA 1977
	DCB	Lawson's Fork Creek, NC	Industrial discharge	32, 12 µg/L	U.S. EPA 1977

DETAILS OF OCCURRENCE OF CHLORINATED BENZENES IN WATER: NORTH AMERICA

COMPOUND	ISOMER	LOCATION	SOURCE	CONCENTRATION	REFERENCE
Dichloro-benzene	DCB	Catawba River	Industrial discharge	33 µg/L	U.S. EPA 1977
	DCB	Kentucky	Industrial waste holding pond	0.9 µg/L	U.S. EPA 1977
	DCB	Ontario	Chemical plant discharge	ND-138 µg/L (DL=0.05 µg/L)	Bonner and Meresz, 1981
	DCB	Sarnia, Ontario	STP - raw sewage final effluent	Trace(0.05-0.1) µg/L 1.0 µg/L	Bonner and Meresz, 1981
	DCB	Wallaceburg. Ontario	STP - raw sewage final effluent	ND-0.1 µg/L ND-4 µg/L (DL=0.05 µg/L)	Bonner and Meresz, 1981
	DCB	Windsor, Ontario (Little River)	STP - raw sewage final effluent	ND-32 µg/L 2-10 µg/L (DL=0.05 µg/L)	Bonner and Meresz, 1981
	DCB	West Windsor, Ontario	STP - raw sewage final effluent	ND µg/L ND-1 µg/L (DL=0.05 µg/L)	Bonner and Meresz, 1981
	1,2-DCB	Miami, FL	Ground water (drinking water supply)	1 µg/L	U.S. EPA, 1977
	1,2-DCB	Kentucky	Ground water	1.2, 71 µg/L	U.S. EPA, 1977
	1,2-DCB	Catawba River	Industrial discharge	690 µg/L	U.S. EPA, 1977
	1,2-DCB	Kentucky	Industrial waste holding pond	15 µg/L	U.S. EPA, 1977

TABLE 3 (Page 4)

DETAILS OF OCCURRENCE OF CHLORINATED BENZENES IN WATER: NORTH AMERICA

COMPOUND	ISOMER	LOCATION	SOURCE	CONCENTRATION	REFERENCE
	1,2-DCB	Big Bigby Creek, Tenn.	Industrial discharge	40 µg/L	U.S. EPA, 1977
	1,2-DCB	Point Loma, California	Municipal discharge	<0.01, 2.2 µg/L	U.S. EPA, 1977
	1,2-DCB	Oxnard, California	Municipal discharge	4.7, 2.3 µg/L	U.S. EPA, 1977
	1,2-DCB	Joint WCP Plant South Calif.	Municipal discharge	5.1, 3.3, 7.3, 12 µg/L	U.S. EPA, 1977
	1,2-DCB	Orange Co. South Calif.	Municipal discharge	1.3, 2.4 µg/L	U.S. EPA, 1977
	1,2-DCB	Los Angeles River	Surface water receiving storm runoff	0.01 µg/L	U.S. EPA, 1977
	1,2-DCB	United States	Highest concentration reported in drinking water	1 µg/L	U.S. EPA, 1975
	1,2-DCB	U.S. National Organic Monitoring Survey	Drinking water supply	Phase *II (0/113)**	U.S. EPA, 1978 (b)
	1,2-DCB	U.S. National Organic Monitoring Survey	Drinking water supply	Phase III 2.5 µg/L(4/110)	U.S. EPA, 1978 (b)
	1,2-DCB	Lake Ontario	Raw water	0.002-0.007 µg/L	Oliver and Nicol, 1982

TABLE 3 (Page 5)

DETAILS OF OCCURRENCE OF CHLORINATED BENZENES IN WATER: NORTH AMERICA

COMPOUND	ISOMER	LOCATION	SOURCE	CONCENTRATION	REFERENCE
	1,2-DCB	Lake Huron	Raw water	ND (DL=0.001 µg/L)	Oliver and Nicol, 1982
	1,2-DCB	Lake Ontario vicinity	Drinking Water supply	ND-0.007 µg/L (DL=0.001 µg/L)	Oliver and Nicol, 1982
	1,2-DCB	Lake Ontario and Grand River vicinity	Effluent discharge	0.006-0.022 µg/L	Oliver and Nicol, 1982
	1,2-DCB	Grand River	Raw water	ND-0.031 µg/L (DL=0.001 µg/L	Oliver and Nicol, 1982
	1,3-DCB	Miami, FL	Ground water (drinking water supply)	0.5 µg/L	U.S. EPA, 1977
	1,3-DCB	United States	Highest concentration reported in drinking water	<3 µg/L	U.S. EPA, 1975
	1,3-DCB	U.S. National Organic Monitoring Survey	Drinking water supply	Phase II (0/113)	U.S. EPA, 1978 (b)
	1,3-DCB	U.S. National Organic Monitoring Survey	Drinking water supply	Phase III 0.10 µg/L (2/110)	U.S. EPA, 1978 (b)
	1,3-DCB	Lake Ontario	Raw water	ND (DL=0.002 µg/L)	Oliver and Nicol, 1982

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DETAILS OF OCCURRENCE OF CHLORINATED BENZENES IN WATER: NORTH AMERICA

COMPOUND	ISOMER	LOCATION	SOURCE	CONCENTRATION	REFERENCE
	1,3-DCB	Lake Huron	Raw water	ND (DL=0.001 µg/L)	Oliver and Nicol, 1982
	1,3-DCB	Lake Ontario vicinity	Drinking water supply	ND (DL=0.001 µg/L)	Oliver and Nicol, 1982
	1,3-DCB	Lake Ontario and Grand River vicinity	Effluent discharge	0.007-0.014 µg/L	Oliver and Nicol, 1982
	1,3-DCB	Grand River	Raw Water	ND-0.004 µg/L (DL=0.001 µg/L)	Oliver and Nicol, 1982
	1,4-DCB	Lake Ontario	Raw water	0.033-0.002 µg/L	Oliver and Nicol, 1982
	1,4-DCB	Lake Huron	Raw water	0.003-0.006 µg/L	Oliver and Nicol, 1982
	1,4-DCB	Lake Ontario vicinity	Drinking water supply	0.008-0.02 µg/L	Oliver and Nicol, 1982
	1,4-DCB	Lake Ontario and Grand River vicinity	Effluent discharge	0.484-0.92 µg/L	Oliver and Nicol, 1982
	1,4-DCB	Grand River	Raw Water	ND-0.042 µg/L (DL = 0.001 µg/L)	Oliver and Nicol, 1982

DETAILS OF OCCURRENCE OF CHLORINATED BENZENES IN WATER:
NORTH AMERICA

COMPOUND	ISOMER	LOCATION	SOURCE	CONCENTRATION	REFERENCE
	1,4-DCB	Miami, FL	Ground water (drinking water supply)	0.5 µg/L	U.S. EPA, 1977
	1,4-DCB	Big Bigby Creek, Tenn.	Industrial discharge	58 µg/L	U.S. EPA, 1977
	1,4-DCB	Point Loma California	Municipal discharge	0.42, 1 µg/L	U.S. EPA, 1977
	1,4-DCB	Oxnard, California	Municipal discharge	9.3, 3.1 µg/L	U.S. EPA, 1977
	1,4-DCB	Joint WCP Plant South Calif.	Municipal discharge	7.6, 7.4, 7.4, 12 µg/L	U.S. EPA, 1977
	1,4-DCB	Orange Co. South Calif.	Municipal discharge	2.8, 4.9 µg/L	U.S. EPA, 1977
	1,4-DCB	Los Angeles River	Surface water receiving storm runoff	0.05 µg/L	U.S. EPA, 1977
	1,4-DCB	United States	Highest concentration reported in drinking water	1 µg/L	U.S. EPA, 1975

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DETAILS OF OCCURRENCE OF CHLORINATED BENZENES IN WATER: NORTH AMERICA

COMPOUND	ISOMER	LOCATION	SOURCE	CONCENTRATION	REFERENCE
	1,4-DCB	U.S. National Organic Monitoring survey	Drinking water supply	Phase I 2.0(2/111) µg/L	U.S. EPA, 1978 (b)
	1,4-DCB	U.S. National Organic Monitoring Survey	Drinking water supply	Phase II 0.14(20/113) µg/L	U.S. EPA, 1978 (b)
	1,4-DCB	U.S. National Organic Monitoring Survey	Drinking water supply	Phase III 0.07(29/110) µg/L	U.S. EPA, 1978 (b)

DETAILS OF OCCURRENCE OF CHLORINATED BENZENES IN WATER: NORTH AMERICA

COMPOUND	ISOMER	LOCATION	SOURCE	CONCENTRATION	REFERENCE
Trichloro-benzene	TCB	Ontario	Chemical plant discharge	ND-80	Bonner and Meresz, 1981
	1,2,3-TCB	Catawba Creek,NC	Municipal discharge	21-46[a] µg/L	U.S. EPA, 1977
	1,2,3-TCB	Lake Ontario	Raw water	0.0001-0.0002 µg/L	Oliver and Nicol, 1982
	1,2,3-TCB	Lake Huron	Raw water	ND (DL=0.0001 µg/L)	Oliver and Nicol, 1982
	1,2,3-TCB	Lake Ontario vicinity	Drinking water supply	0.0001-0.001 µg/L	Oliver and Nicol, 1982
	1,2,3-TCB	Lake Ontario and Grand River vicinity	Effluent discharge	0.002-0.003 µg/L	Oliver and Nicol, 1982
	1,2,3-TCB	Grand River	Raw Water	ND=0.0007 µg/L (DL=0.0001 µg/L)	Oliver and Nicol, 1982
	1,2,4-TCB	Catawba Creek,NC	Industrial discharge	12[a] µg/L	U.S. EPA, 1977
	1,2,4-TCB	Chattanooga Creek, NC	Industrial discharge	500[b] µg/L	U.S. EPA, 1977
	1,2,4-TCB	Joint Water Pollution Control Plant (JPWCP)	Municipal waste water	6.0; 1.8[a] µg/L	U.S. EPA, 1977
	1,2,4-TCB	Orange County Sewage Department (OCSD)	Municipal waste water	0.30[a] µg/L	U.S. EPA, 1977
	1,2,4-TCB	Port Loma Sewage Treatment Plant (PLSTP)	Municipal waste water	0.23; <0.01[a] µg/L	U.S. EPA, 1977

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DETAILS OF OCCURRENCE OF CHLORINATED BENZENES IN WATER: NORTH AMERICA

COMPOUND	ISOMER	LOCATION	SOURCE	CONCENTRATION	REFERENCE
	1,2,4-TCB	Oxnard, CA Sewage Treatment Plant (OSTP)	Municipal, waste water	0.9; 0.25[c] µg/L	U.S. EPA, 1977
	1,2,4-TCB	Los Angeles River	Surface run off	0.007[d] µg/L	U.S. EPA, 1977
	1,2,4-TCB	Lake Ontario	Raw water	0.0003-0.0001 µg/L	Oliver and Nicol, 1982
	1,2,4-TCB	Lake Huron	Raw water	0.0001-0.0004 µg/L	Oliver and Nicol, 1982
	1,2,4-TCB	Lake Ontario vicinity	Drinking water supply	0.001-0.004 µg/L	Oliver and Nicol, 1982
	1,2,4-TCB	Lake Ontario and Grand River vicinity	Effluent discharge	0.005-0.018 µg/L	Oliver and Nicol, 1982
	1,2,4-TCB	Grand River	Raw Water	ND-0.008 µg/L (DL=0.0001 µg/L)	Oliver and Nicol, 1982
	1,3,5-TCB	Holston River, TN	Industrial discharge	26 µg/L	U.S. EPA, 1977
	1,3,5-TCB	Joint Water Pollution Control Plant (JPWCP)	Municipal waste water	0.2; 0.8[c] µg/L	U.S. EPA, 1977
	1,3,5-TCB	Orange County Sewage Department (OCSD)	Municipal waste water	0.2 µg/L	U.S. EPA, 1977

DETAILS OF OCCURRENCE OF CHLORINATED BENZENES IN WATER: NORTH AMERICA

COMPOUND	ISOMER	LOCATION	SOURCE	CONCENTRATION	REFERENCE
	1,3,5-TCB	Port Loma Sewage Treatment Plant (PLSTP)	Municipal waste water	0.02; <0.01[c] µg/L	U.S. EPA, 1977
	1,3,5-TCB	Oxnard, CA Sewage Treatment Plant (OSTP)	Municipal waste water	0.4; <0.01[c] µg/L	U.S. EPA, 1977
	1,3,5-TCB	Los Angeles River	Surface run off	0.006[c] µg/L	U.S. EPA, 1977
	1,3,5-DCB	Lake Ontario	Raw water	ND-0.0004 µg/L (DL=0.0001 µg/L)	Oliver and Nicol, 1982
	1,3,5-DCB	Lake Huron	Raw water	ND (DL=0.0001 µg/L)	Oliver and Nicol, 1982
	1,3,5-DCB	Lake Ontario vicinity	Drinking water supply	ND (DL=0.0001 µg/L)	Oliver and Nicol, 1982
	1,3,5-DCB	Lake Ontario and Grand River vicinity	Effluent discharge	0.0001-0.0005 µg/L	Oliver and Nicol, 1982
	1,3,5-DCB	Grand River	Raw Water	ND (DL=0.0001 µg/L)	Oliver and Nicol, 1982

DETAILS OF OCCURRENCE OF CHLORINATED BENZENES IN WATER: NORTH AMERICA

COMPOUND	ISOMER	LOCATION	SOURCE	CONCENTRATION	REFERENCE
Tetrachloro-benzene	1,2,3,4-TeCB	Lake Ontario	Raw water	ND - 0.0005 µg/L	Oliver and Nicol, 1982
	1,2,3,4-TeCB	Lake Huron	Raw water	0.00004-0.00005 µg/L	Oliver and Nicol, 1982
	1,2,3,4-TeCB	Lake Ontario vicinity	Drinking water supply	0.0001-0.0004 µg/L	Oliver and Nicol, 1982
	1,2,3,4-TeCB	Lake Ontario and Grand River vicinity	Effluent discharge	0.0002-0.003 µg/L	Oliver and Nicol, 1982
	1,2,3,4-TeCB	Grand River	Raw Water	ND-0.0002 µg/L (DL=0.00005 µg/L)	Oliver and Nicol, 1982
	1,2,3,5-TeCB	Lake Ontario	Raw water	ND (DL=0.00005 µg/L)	Oliver and Nicol, 1982
	1,2,3,5-TeCB	Lake Huron	Raw water	ND (DL=0.00005 µg/L)	Oliver and Nicol, 1982
	1,2,3,5-TeCB	Lake Ontario vicinity	Drinking water supply	ND (DL=0.00005 µg/L)	Oliver and Nicol, 1982
	1,2,3,5-TeCB	Lake Ontario and Grand River vicinity	Effluent discharge	0.0001-0.001 µg/L	Oliver and Nicol, 1982
	1,2,3,5-TeCB	Grand River	Raw Water	ND (DL=0.00005 µg/L)	Oliver and Nicol, 1982

DETAILS OF OCCURRENCE OF CHLORINATED BENZENES IN WATER: NORTH AMERICA

COMPOUND	ISOMER	LOCATION	SOURCE	CONCENTRATION	REFERENCE
Tetrachloro-benzene	1,2,4,5-TeCB	Lake Ontario	Raw water	ND-0.0003 µg/L (DL=0.00005 µg/L)	Oliver and Nicol, 1982
	1,2,4,5-TeCB	Lake Huron	Raw water	ND (DL=0.00005 µg/L)	Oliver and Nicol, 1982
	1,2,4,5-TeCB	Lake Ontario vicinity	Drinking water supply	ND-0.0003 µg/L (DL=0.00005 µg/L)	Oliver and Nicol, 1982
	1,2,4,5-TeCB	Lake Ontario and Grand River vicinity	Effluent discharge	0.0003-0.002 µg/L	Oliver and Nicol, 1982
	1,2,4,5-TeCB	Grand River	Raw Water	ND (DL=0.00005 µg/L)	Oliver and Nicol, 1982
Pentachloro-benzene	QCB	Lake Ontario	Raw water	ND-0.0006 µg/L (DL=0.00001 µg/L)	Oliver and Nicol, 1982
	QCB	Lake Huron	Raw water	0.00003-0.00004 µg/L	Oliver and Nicol, 1982
	QCB	Lake Ontario vicinity	Drinking water supply	0.00003-0.00005 µg/L	Oliver and Nicol, 1982
	QCB	Lake Ontario and Grand River vicinity	Effluent discharge	0.0005-0.001 µg/L	Oliver and Nicol, 1982
	QCB	Grand River	Raw Water	ND-0.0001 µg/L (DL=0.00001 µg/L)	Oliver and Nicol, 1982
	QCB	Southern California	Waste water treatment plant	detected	Burlingame, 1977

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DETAILS OF OCCURRENCE OF CHLORINATED BENZENES IN WATER: NORTH AMERICA

COMPOUND	ISOMER	LOCATION	SOURCE	CONCENTRATION	REFERENCE
Hexachloro-benzene	HCB	Louisiana	Lower Mississippi River in vicinity of industrialized region	Usually below 2 µg/L	Laska et al., 1976
	HCB	Louisiana	Landfill pond water	4.8-74.9 µg/L	Laseter et al., 1976
	HCB	Louisiana	Stream adjacent to industrial plant	0.1-72.8 µg/L	Laseter et al., 1976
	HCB	Louisiana	Drinking water supplies (3 locations)	0.006-0.01µg/L	U.S. EPA, 1975
	HCB	Louisiana	Finished drinking water (2 locations)	0.004-0.006 µg/L	U.S. EPA, 1975
	HCB	Lake Superior	Rain water	0.003 µg/L	Strachan and Glass, 1978
	HCB	Lake Ontario	Surface water	0.00001-0.0001 µg/L	Oliver and Nicol, 1982
	HCB	Lake Huron	Raw water	0.00002-0.0001 µg/L	Oliver and Nicol, 1982
	HCB	Lake Ontario vicinity	Drinking water supply	0.00006-0.0002 µg/L	Oliver and Nicol, 1982
	HCB	Lake Ontario and Grand River vicinity	Effluent discharge	0.001-0.002 µg/L	Oliver and Nicol, 1982
	HCB	Grand River	Raw Water	0.00002-0.0001 µg/L	Oliver and Nicol, 1982
	HCB	Great Lakes	Snowfall	ND (DL=0.0002 µg/L)	Strachan and Glass, 1978

DETAILS OF OCCURRENCE OF CHLORINATED BENZENES IN WATER: NORTH AMERICA

COMPOUND	ISOMER	LOCATION	SOURCE	CONCENTRATION	REFERENCE
Hexachloro-benzene	HCB	Great Lakes	Rainfall'	ND-0.0028 µg/L (DL=0.002 µg/L)	Strachan and Glass, 1978
	HCB	Great Lakes	Rainfall	0.001-0.004 µg/L	Eisenreich et al., 1981
	HCB	Ontario	Chemical effluent	0.005-0.007 µg/L	Gilbertson, 1978

Notes: ND - not detected
DL - detection limit

[a]Summer; [b]Spring; [c]Summer, Fall; [d]Winter; [e]Fall

* Phase I: samples shipped and stored at 2 to 8°C for 1 to 2 weeks prior to analyses;
Phase II: samples held at 20 to 25°C for 3 to 6 weeks prior to analyses permitting reactions to proceed to end points (terminal values);
Phase III: samples processed with or without chlorine reducing agent.
In these data samples were processed without quenching additive, so were permitted to react to terminal values.

** (Number of positive analyses/number of analyses)

TABLE 3 (Page 16)

DETAILS OF OCCURRENCE OF CHLORINATED BENZENES IN WATER: OUTSIDE NORTH AMERICA

COMPOUND	ISOMER	LOCATION	SOURCE	CONCENTRATION	REFERENCE
Dichloro-benzene	1,4-DCB	Glatt River, Switzerland	River water (ave. n = 8)	0.28 µg/L	Schneider et al., 1981
	1,4-DCB	England	Groundwater	ND-0.08 (±0.22) µg/L	Fielding et al., 1981
	1,4-DCB	England	River	ND DL=0.01 µg/L	Fielding et al., 1981
	1,4-DCB	England	Rain	0.01(±0.005) µg/L	Fielding et al., 1981
Hexachloro-benzene	HCB	Italy	Various surface waters	Ave: 0.0025 µg/L Range:(ND-0.0251 µg/L)	Leoni and D'arca, 1976
	HCB	Holland	Rhine River	Ave: 0.130 µg/L Range:(0.01-0.52 µg/L)	Greve, 1972
	HCB	Holland	Groundwater	Highest observed value = 10 µg/L	Zoeteman et al., 1981

Notes: ND - not detectable
DL - detection limit
Ave. - average

TABLE 4

SUMMARY OF OCCURRENCE OF
CHLORINATED BENZENES IN SEDIMENT

MPOUND	ISOMER	GREAT LAKES	CANADA OTHER THAN GREAT LAKES	US OTHER THAN GREAT LAKES
hlorobenzene	1,2-DCB	ND-56 µg/kg** (DL= 5 µg/kg)	-	-
	1,3-DCB	ND-250 µg/kg** (DL= 5 µg/kg)	-	-
	1,4-DCB	ND-210 µg/kg** (DL= 5 µg/kg)	-	-
chlorobenzene	1,2,3-TCB	ND-16 µg/kg** (DL= 0.4 µg/kg)	-	-
	1,2,4-TCB	trace-220 µg/kg**	-	-
	1,3,5-TCB	ND-250 µg/kg** (DL= 0.4 µg/kg)	-	-
rachlorobenzenes	1,2,3,4-TeCB	ND-145 µg/kg** (DL= 0.2 µg/kg)	-	-
	1,2,3,5-TeCB	ND-13 µg/kg** (DL= 0.2 µg/kg)	-	-
	1,2,4,5-TeCB	ND-160 µg/kg** (DL= 0.2 µg/kg)	-	-
ntachlorobenzenes	QCB	ND-115 µg/kg** (DL= 0.05 µg/kg)	2-10 µg/kg (Economy R., N.S.)	-
kachlorobenzenes	HCB	0.02-320 µg/kg**	ND-0.033 µg/kg** (Rattray Marsh) 2-10 µg/kg (Economy R., N.S.)	<.7-400 µg/kg* (Mississippi River)

tes: ND - not detectable
DL - detection limit
* - dry weight basis
** - wet weight basis

TABLE 4 (Page 2)

DETAILS OF OCCURRENCE OF CHLORINATED BENZENES IN SEDIMENT

COMPOUND	ISOMER	LOCATION	CONCENTRATION	REFERENCE
Dichlorobenzene	DCB	St. Clair River	Identified	Bonner and Meresz, 1981
Dichlorobenzene	1,2-DCB	Lake Superior	ND - 1 µg/kg**	Oliver and Nicol, 1982
		Lake Huron	ND - 56 µg/kg**	Oliver and Nicol, 1982
		Lake Erie	1 - 4 µg/kg**	Oliver and Nicol, 1982
		Lake Ontario	4 - 27 µg/kg**	Oliver and Nicol, 1982
Dichlorobenzene	1,3-DCB	Lake Superior, surficial sediments	ND-7 µg/kg**	Oliver and Nicol, 1982
		Lake Huron, surficial sediments	ND-14 µg/kg**	Oliver and Nicol, 1982
		Lake Erie, surficial sediments	1-9 µg/kg**	Oliver and Nicol, 1982
		Lake Ontario, surficial sediments	15-250 µg/kg**	Oliver and Nicol, 1982
Dichlorobenzene	1,4-DCB	Lake Superior, surficial sediments	ND-9 µg/kg**	Oliver and Nicol, 1982
		Lake Huron, surficial sediments	2-100 µg/kg**	Oliver and Nicol, 1982
		Lake Erie, surficial sediments	3-20 µg/kg**	Oliver and Nicol, 1982
		Lake Ontario, surficial sediments	22-210 µg/kg**	Oliver and Nicol, 1982
Trichlorobenzene	1,2,3-TCB	Lake Superior	ND-1 µg/kg**	Oliver and Nicol, 1982
		Lake Huron	0.1-1 µg/kg**	Oliver and Nicol, 1982
		Lake Erie	0.1-1 µg/kg**	Oliver and Nicol, 1982
		Lake Ontario	1-16 µg/kg**	Oliver and Nicol, 1982

TABLE 4 (Page 3)

DETAILS OF OCCURRENCE OF CHLORINATED BENZENES IN SEDIMENT

COMPOUND	ISOMER	LOCATION	CONCENTRATION	REFERENCE
Trichlorobenzene	TCB	St. Clair River	Identified	Bonner and Meresz, 1981
Trichlorobenzene	1,2,4-TCB	Lake Superior	0.1-4 µg/kg**	Oliver and Nicol, 1982
		Lake Huron	1-26 µg/kg**	Oliver and Nicol, 1982
		Lake Erie	1-9 µg/kg**	Oliver and Nicol, 1982
		Lake Ontario	20-220 µg/kg**	Oliver and Nicol, 1982
Trichlorobenzene	1,3,5-TCB	Lake Superior	ND - 0.4 µg/kg**	Oliver and Nicol, 1982
		Lake Huron	ND - 4 µg/kg**	Oliver and Nicol, 1982
		Lake Erie	0.1 - 5 µg/kg**	Oliver and Nicol, 1982
		Lake Ontario	7 - 250 µg/kg**	Oliver and Nicol, 1982
Tetrachlorobenzene	1,2,3,4 -TeCB	Lake Superior	ND-1 µg/kg**	Oliver and Nicol, 1982
		Lake Huron	0.2-8 µg/kg**	Oliver and Nicol, 1982
		Lake Erie	0.2-2 µg/kg**	Oliver and Nicol, 1982
		Lake Ontario	11-86 µg/kg**	Oliver and Nicol, 1982
Tetrachlorobenzene	1,2,3,5 - TeCB	Lake Superior	ND-0.3 µg/kg**	Oliver and Nicol, 1982
		Lake Huron	0.1-0.7 µg/kg**	Oliver and Nicol, 1982
		Lake Erie	0.1-0.9 µg/kg**	Oliver and Nicol, 1982
		Lake Ontario	1-13 µg/kg**	Oliver and Nicol, 1982

TABLE 4 (Page 4)

DETAILS OF OCCURRENCE OF CHLORINATED BENZENES IN SEDIMENT

COMPOUND	ISOMER	LOCATION	CONCENTRATION	REFERENCE
Tetrachlorobenzene	1,2,4,5 - TeCB	Lake Superior	ND-1 µg/kg**	Oliver and Nicol, 1982
		Lake Huron	0.2- 9 µg/kg**	Oliver and Nicol, 1982
		Lake Erie	0.3-4 µg/kg**	Oliver and Nicol, 1982
		Lake Ontario	6-160 µg/kg**	Oliver and Nicol, 1982
		Economy River, Nova Scotia	2-10 µg/kg**	Wilson et al., 1979
Pentachlorobenzene	QCB	Lake Superior	ND-0.3 µg/kg**	Oliver and Nicol, 1982
		Lake Huron	0.2-3 µg/kg**	Oliver and Nicol, 1982
		Lake Erie	0.3-3 µg/kg**	Oliver and Nicol, 1982
		Lake Ontario	3-84 µg/kg**	Oliver and Nicol, 1982
Hexachlorobenzene	HCB	Lake Superior	0.02-0.7 µg/kg**	Oliver and Nicol, 1982
		Lake Huron	0.4-5 µg/kg**	Oliver and Nicol, 1982
		Lake Erie	0.7-12 µg/kg**	Oliver and Nicol, 1982
		Lake Ontario	9-320 µg/kg**	Oliver and Nicol, 1982
		Rattray Marsh (Lake Ontario shoreline) Mississauga, Ontario	ND-0.033 µg/kg*	Glooschenko et al., 1981
		Economy River	2-10 µg/kg	Wilson et al., 1980
		Mississippi River	<.7-400 µg/kg***	Laska et al., 1976

ND - not detectable, 5 µg/kg DCB, 0.4 µg/kg TCB, 0.2 µg/kg TeCB, 0.05 µg/kg QCB and HCB
* - ND level not defined
** - wet weight basis
*** - dry weight basis

TABLE 5

SUMMARY OF OCCURRENCE OF
CHLORINATED BENZENES IN AQUATIC BIOTA

COMPOUND	ISOMER	GREAT LAKES	CANADA OTHER OTHER THAN GREAT LAKES	USA OTHER THAN GREAT LAKES	OUTSIDE NORTH AMERICA
Dichlorobenzene	1,2-DCB	0.3-1 µg/kg			trace - 1200 µg/kg
	1,3-DCB	0.3-3 µg/kg			
	1,4-DCB	ND-4 µg/kg (DL= 5 µg/kg)			trace - 45 µg/kg
Trichlorobenzene	1,2,3-TCB	0.1-13 µg/kg		NQ-20 µg/kg	8-48 µg/kg
	1,2,4-TCB	0.5-36.0 µg/kg		NQ-124 µg/kg	1-15 µg/kg
	1,3,5-TCB	0.1-4 µg/kg		2.1-75 µg/kg	1-5 µg/kg
Tetrachlorobenzene	1,2,3,4-TeCB	0.3-220 µg/kg		1.3-205 µg/kg	trace - 5 µg/kg
	1,2,3,5-TeCB	0.05-43 µg/kg			
	1,2,4,5-TeCB	0.2-12 µg/kg		7-1150 µg/kg	
Pentachlorobenzene	QCB	0.6-24 µg/kg		19-789 µg/kg	6-90 µg/kg
Hexachlorobenzene	HCB	ND-230 µg/kg (DL=5 µg/kg)	ND-55 µg/kg	<5-48699 µg/kg	56-349 µg/kg

Notes: ND, not detected
DL, detection limit
NQ, detected but not quantified

TABLE 5 (Page 2)

DETAILS OF OCCURRENCE OF CHLORINATED BENZENES IN AQUATIC BIOTA: NORTH AMERICA

COMPOUND	ISOMER	LOCATION	SPECIES	CONCENTRATION	REFERENCE
Dichloro-benzene	1,2-DCB	Lake Superior	Salvelinus namaycush	0.3 µg/kg	Oliver and Nicol, 1982
		Lake Huron	Salvelinus namaycush	1 µg/kg	Oliver and Nicol, 1982
		Lake Erie	Salmo gairdneri	1 µg/kg	Oliver and Nicol, 1982
		Lake Ontario	Salvelinus namaycush	1 µg/kg	Oliver and Nicol, 1982
		Lake Ontario	Salvelinus namaycush	1 µg/kg	Oliver and Nicol, 1982
	1,3-DCB	Lake Superior	Salvelinus namaycush	0.6 µg/kg	Oliver and Nicol, 1982
		Lake Huron	Salvelinus namaycush	0.6 µg/kg	Oliver and Nicol, 1982
		Lake Erie	Salmo gairdneri	0.3 µg/kg	Oliver and Nicol, 1982
		Lake Ontario	Salvelinus namaycush	3 µg/kg	Oliver and Nicol, 1982
		Lake Ontario	Salvelinus namaycush	2 µg/kg	Oliver and Nicol, 1982
	1,4-DCB	Lake Superior	Salvelinus namaycush	N.D. (D.L.= 5 µg/kg)	Oliver and Nicol, 1982
		Lake Huron	Salvelinus namaycush	1 µg/kg	Oliver and Nicol, 1982
		Lake Erie	Salmo gairdneri	4 µg/kg	Oliver and Nicol, 1982
		Lake Ontario	Salvelinus namaycush	4 µg/kg	Oliver and Nicol, 1982
		Lake Ontario	Salvelinus namaycush	2 µg/kg	Oliver and Nicol, 1982

DETAILS OF OCCURRENCE OF CHLORINATED BENZENES IN AQUATIC BIOTA: NORTH AMERICA

COMPOUND	ISOMER	LOCATION	SPECIES	CONCENTRATION	REFERENCE
Trichloro-benzene	1,2,3-TCB	Lake Superior	Salvelinus namaycush	0.1 µg/kg	Oliver and Nicol, 1982
		Lake Huron	Salvelinus namaycush	0.2 µg/kg	Oliver and Nicol, 1982
		Lake Erie	Salmo gairdneri	0.1 µg/kg	Oliver and Nicol, 1982
		Lake Ontario	Salvelinus namaycush	1 µg/kg	Oliver and Nicol, 1982
		Lake Ontario	Salvelinus namaycush	0.2 µg/kg	Oliver and Nicol, 1982
		Lake Ontario	fish	present	Kaiser, 1982
		Lake Ontario	Salmonid species	NQ-9.5 µg/kg	Kuehl et al., 1981
		Great Lakes other than Lake Ontario	Various fish species	ND (DL=1 µg/kg)	Kuehl et at., 1981
		Ashtabula R. and Fields Br. Ohio	Composite fish samples	NQ-20 µg/kg	Kuehl et al., 1981
	1,2,4-TCB	Lake Superior	Salvelinus namaycush	0.6 µg/L	Oliver and Nicol, 1982
		Lake Huron	Salvelinus namaycush	1 µg/L	Oliver and Nicol, 1982
		Lake Erie	Salmo gairdneri	0.5 µg/L	Oliver and Nicol, 1982
		Lake Ontario	Salvelinus namaycush	5 µg/L	Oliver and Nicol, 1982
		Lake Ontario	Salvelinus namaycush	2 µg/L	Oliver and Nicol, 1982

TABLE 5 (Page 4)

DETAILS OF OCCURRENCE OF CHLORINATED BENZENES IN AQUATIC BIOTA: NORTH AMERICA

COMPOUND	ISOMER	LOCATION	SPECIES	CONCENTRATION	REFERENCE
	1,2,4-TCB	Lake Ontario	Salmonid species	2.4-3.6 µg/kg	Kuehl et al., 1981
		Lake Superior	Salvelinus namaycush	ND-1.2 µg/kg (DL=1 µg/kg)	Kuehl et al., 1981
		Lake Michigan	Salvelinus namaycush	2.0 µg/kg	Kuehl et al., 1981
		Lake Huron	Various fish species	ND-1.4 µg/kg (DL=1 µg/kg)	Kuehl et al., 1981
		Ashtabula R. and Fields Br., Ohio	Various fish species	NQ-124 µg/kg	Kuehl et al., 1981
	1,3,5-TCB	Lake Superior	Salvelinus namaycush	0.6 µg/kg	Oliver and Nicol, 1982
		Lake Huron	Salvelinus namaycush	0.3 µg/kg	Oliver and Nicol, 1982
		Lake Erie	Salmo gairdneri	0.1 µg/kg	Oliver and Nicol, 1982
		Lake Ontario	Salvelinus namaycush	4 µg/kg	Oliver and Nicol, 1982
		Lake Ontario	Salvelinus namaycush	4 µg/kg	Oliver and Nicol, 1982
		Lake Ontario	Salmonid species	detected	Kuehl et al., 1981
		Great Lakes other than Lake Ontario	Various fish species	ND (DL=1 µg/kg)	Kuehl et at., 1981
		Ashtabula R. and Fields Br. Ohio	Various fish species	2.1-75 µg/kg	Kuehl et al., 1981

TABLE 5 (Page 5)

DETAILS OF OCCURRENCE OF CHLORINATED BENZENES IN AQUATIC BIOTA: NORTH AMERICA

COMPOUND	ISOMER	LOCATION	SPECIES	CONCENTRATION	REFERENCE
Tetrachloro-benzene	1,2,3,4-TeCB	Lake Superior	Salvelinus namaycush	0.5 µg/kg	Oliver and Nicol, 1982
		Lake Huron	Salvelinus namaycush	2 µg/kg	Oliver and Nicol, 1982
		Lake Erie	Salmo gairdneri	0.3 µg/kg	Oliver and Nicol, 1982
		Lake Ontario	Salvelinus namaycush	12 µg/kg	Oliver and Nicol, 1982
		Lake Ontario	Salvelinus namaycush	4 µg/kg	Oliver and Nicol, 1982
		Lake Ontario	Savelinus namaycush	5-17 µg/kg	Kuehl et al., 1981
		Great Lakes other than Lake Ontario	Various fish species	ND (DL=5 µg/kg)	Kuehl et at., 1981
		Ashtabula R. and Fields Br. Ohio	Various fish species	1.3-205 µg/kg	Kuehl et al., 1981
	1,2,3,5-TeCB	Lake Superior	Salvelinus namaycush	0.1 µg/kg	Oliver and Nicol, 1982
		Lake Huron	Salvelinus namaycush	0.2 µg/kg	Oliver and Nicol, 1982
		Lake Erie	Salvelinus namaycush	0.05 µg/kg	Oliver and Nicol, 1982
		Lake Ontario	Salvelinus namaycush	1 µg/kg	Oliver and Nicol, 1982
		Lake Ontario	Salvelinus namaycush	0.5 µg/kg	Oliver and Nicol, 1982

TABLE 5 (Page 6)

DETAILS OF OCCURRENCE OF CHLORINATED BENZENES IN AQUATIC BIOTA: NORTH AMERICA

COMPOUND	ISOMER	LOCATION	SPECIES	CONCENTRATION	REFERENCE
	1,2,4,5-TeCB	Lake Superior	Salvelinus namaycush	0.3 µg/kg	Oliver and Nicol, 1982
		Lake Huron	Salvelinus namaycush	1 µg/kg	Oliver and Nicol, 1982
		Lake Erie	Salmo gairdneri	0.2 µg/kg	Oliver and Nicol, 1982
		Lake Ontario	Salvelinus namaycush	5 µg/kg	Oliver and Nicol, 1982
		Lake Ontario	Salvelinus namaycush	2 µg/kg	Oliver and Nicol, 1982
		Lake Ontario	Savelinus namaycush	2.5-12 µg/kg	Kuehl et al., 1981
		Great Lakes other than Lake Ontario	Various fish species	ND (DL=5 µg/kg)	Kuehl et al., 1981
		Ashtabula R. and Fields Br., Ohio	Various fish species	7-1150 µg/kg	Kuehl et al., 1981
	not determined	Lake Ontario	fish	present	Kaiser, 1977
Pentachloro-benzene	QCB	Lake Superior	Salvelinus namaycush	0.7 µg/kg	Oliver and Nicol, 1982
		Lake Huron	Salvelinus namaycush	2 µg/kg	Oliver and Nicol, 1982
		Lake Erie	Salmo gairdneri	0.6 µg/kg	Oliver and Nicol, 1982
		Lake Ontario	Salvelinus namaycush	16 µg/kg	Oliver and Nicol, 1982
		Lake Ontario	Salvelinus namaycush	8 µg/kg	Oliver and Nicol, 1982

TABLE 5 (Page 7)

DETAILS OF OCCURRENCE OF CHLORINATED BENZENES IN AQUATIC BIOTA: NORTH AMERICA

COMPOUND	ISOMER	LOCATION	SPECIES	CONCENTRATION	REFERENCE
Pentachloro benzene	QCB	Lake Ontario	Savelinus namaycush	9.1-17 µg/kg	Kuehl et al., 1981
		Great Lakes other than Lake Ontario	Various fish species	ND (DL=5 µg/kg)	Kuehl et al., 1981
		Ashtabula R. and Fields Br., Ohio	Various fish species	19-789 µg/kg	Kuehl et al., 1981
Hexachloro-benzene	HCB	Lake Superior	Salvelinus namaycush	13 µg/kg	Oliver and Nicol, 1982
		Lake Huron	Salvelinus namaycush	25 µg/kg	Oliver and Nicol, 1982
		Lake Erie	Salmo gairdneri	8 µg/kg	Oliver and Nicol, 1982
		Lake Ontario	Salvelinus namaycush	127 µg/kg	Oliver and Nicol, 1982
		Lake Ontario	Salvelinus namaycush	61 µg/kg	Oliver and Nicol, 1982
		Lake Ontario	fish	present	Kaiser, 1982
		Lake Ontario	Salvelinus namaycush	115-230 µg/kg	Kuehl et al., 1981
		Lake Superior	Salvelinus namaycush	ND-6.5 µg/kg (DL=5 µg/kg)	Kuehl et al., 1981
		Lake Michigan	Savelinus namaycush	6.3 µg/kg	Kuehl et al., 1981
		Lake Huron	Various fish species	NQ-11.5 µg/kg	Kuehl et al., 1981
		Ashtabula R. and Fields Br., Ohio	Various fish species	48-2210 µg/kg	Kuehl et al., 1981

TABLE 5 (Page 8)

DETAILS OF OCCURRENCE OF CHLORINATED BENZENES IN AQUATIC BIOTA: NORTH AMERICA

COMPOUND	ISOMER	LOCATION	SPECIES	CONCENTRATION	REFERENCE
Hexachloro-benzene	HCB	Lake Ontario	alewives and smelt muscle *Alosa pseudoharengus* and *Osmerus mordax*	24 µg/kg	Norstrom et al., 1978
		Lake Ontario	coho salmon muscle *Oncorhynchus kisutch*	97 µg/kg muscle	Norstrom et al., 1978
		Lake Ontario	coho salmon muscle *Oncorhynchus kisutch*	65 µg/kg liver	Norstrom et al., 1978
		Lake Ontario	salmonids	16-125 µg/kg	Niimi, 1979
		Lake Ontario	Various fish species	10-140 µg/kg	Gilbertson, 1978
		Hamilton Beach, Lake Ontario	sea lamprey *Petromyzon marinus*	90 µg/kg	Kaiser and Valdmanis, 1978
		Northern sections of Lakes Huron	sea lamprey *Petromyzon marinus*	8±4 µg/kg sig = .01	Kaiser, 1982
		Northern sections of Lakes Huron	lake whitefish *Coregonus clupeaformis*	18±9 µg/kg sig = .01	Kaiser, 1982
		Hamilton Harbour	Various fish species	10-230 µg/kg	MOE, 1974
		Lake Superior	lake trout *Salvelinus namaycush*	BQT-5 µg/kg	Swain 1978
		Lake Superior	lake whitefish *Coregonus culpeaformis*	3-5 µg/kg	Swain, 1978

DETAILS OF OCCURRENCE OF CHLORINATED BENZENES IN AQUATIC BIOTA: NORTH AMERICA

COMPOUND	ISOMER	LOCATION	SPECIES	CONCENTRATION	REFERENCE
Hexachloro-benzene	HCB	Siskiwit Lake, Isle Royale	lake trout *Salvelinus namaycush*	5 µg/kg	Swain, 1978
		Siskiwit Lake, Isle Royale	lake whitefish *Coregonus culpeaformis*	BQT	Swain, 1978
		United States Watersheds near Great Lakes	channel catfish *Ictalurus punctatus*	<5-7 µg/kg	Veith et al., 1979(b)
		Sandusky R., Ohio	freshwater drum *Apiodinotus grunniens*	<5 µg/kg	Veith et al., 1979(b)
		Lake Ontario, near Oswega, N.Y.	brown trout *Salmo trutta*	87 µg/kg	Veith et al., 1979(b)
		Cottaragaus Creek New York	coho salmon *Oncorhynchus kisutch*	7 µg/kg	Veith et al., 1979(b)
		Ashtabula River, Ohio	Various fish species	447 µg/kg (highest value)	Veith et al., 1979(b)
		Lake Saint Clair	several fish species	<1 - 80 µg/kg	Frank et al., 1978 (b)
		Lake Huron	several fish species	ND-30 µg/kg	Frank et al., 1978 (a)
		Cold Lake area Alberta	several fish species	ND-55 µg/kg (D.L. = 1 µg/kg)	Tsui, 1981
		Mississipi River	Mosquito fish *Gambusia affinis*	72-380 µg/kg	Laska et al., 1974

DETAILS OF OCCURRENCE OF CHLORINATED BENZENES IN AQUATIC BIOTA: NORTH AMERICA

COMPOUND	ISOMER	LOCATION	SPECIES	CONCENTRATION	REFERENCE
Hexachloro-benzene	HCB	Lower Mississippi R., Louisiana	Plankton, undefined	561 µg/kg	Laseter et al., 1976
		Lower Mississippi R., Louisiana	Chara sp.	563 µg/kg	Laseter et al., 1976
		Lower Mississippi R., Louisiana	Najas sp.	147 µg/kg	Laseter et al., 1976
		Lower Mississippi R., Louisiana	Eleocharis sp.	423 µg/kg	Laseter et al., 1976
		Lower Mississippi R., Louisiana	Physa sp.	294 µg/kg	Laseter et al., 1976
		Lower Mississippi R., Louisiana	Procambarus sp.	48669 µg/kg**	Laseter et al., 1976
		Lower Mississippi R., Louisiana	Anisoptera	4699 µg/kg**	Laseter et al., 1976
		United States	inland fish species except carp	ND-20000 µg/kg	Johnson et al., 1974
		United States	carp	6-62000 µg/kg**	Johnson et al., 1974
		Wabash River, Indiana	composite fish samples	30 µg/kg	Kuehl et al., 1980
		Ashtabula River, Ohio	composite fish samples	3140 µg/kg	Kuehl et al., 1980
		Altamaha River, Darien, Georgia	composite fish samples	11600 µg/kg	Veith et al., 1979

DETAILS OF OCCURRENCE OF CHLORINATED BENZENES IN AQUATIC BIOTA: NORTH AMERICA

COMPOUND	ISOMER	LOCATION	SPECIES	CONCENTRATION	REFERENCE
Hexachloro-benzene		United States Watersheds near Great Lakes	carp *Cyprinus carpio*	<5-37 µg/kg	Veith et al., 1979(b)
		United States Watersheds near Great Lakes	Various fish species	5-447 µg/kg	Veith et al., 1981

DETAILS OF OCCURRENCE OF CHLORINATED BENZENES IN AQUATIC BIOTA: OUTSIDE NORTH AMERICA

COMPOUND	ISOMER	LOCATION	SPECIES	CONCENTRATION	REFERENCE
Dichloro-benzene	1,2-DCB	Kokra R., Yugoslavia	brown trout *Salmo trutta*	120 µg/kg	Jan and Malnersic, 1980
		Drava R. after Maribor Yugoslavia	nace *Chondrostoma nasus*	1140 µg/kg	Jan and Malnersic, 1980
		Mura R. Basin, Slovenia Yugoslavia	nace *Chondrostoma nasus*	1200 µg/kg	Jan and Malnersic, 1980
		Sava R. after Ljubljama Yugoslavia	brown trout *Salmo trutta*	1100 µg/kg	Jan and Malnersic, 1980
		Sava R. after Ljubljama Yugoslavia	whiting *Leuciscuso cephalus*	trace	Jan and Malnersic, 1980
	1,4-DCB	Kokra R., Yugoslavia	brown trout *Salmo trutta*	trace	Jan and Malnersic, 1980
		Drava R. after Maribor Yugoslavia	nace *Chondrostoma nasus*	450 µg/kg	Jan and Malnersic, 1980
		Mura R. Basin, Yugoslavia	nace *Chondrostoma nasus*	350 µg/kg	Jan and Malnersic, 1980

DETAILS OF OCCURRENCE OF CHLORINATED BENZENES IN AQUATIC BIOTA: OUTSIDE NORTH AMERICA

COMPOUND	ISOMER	LOCATION	SPECIES	CONCENTRATION	REFERENCE
Dichloro-benzene	1,4-DCB	Sava R. after Ljubljama, Yugoslavia	nace *Chondrostoma nasus*	220 µg/kg	Jan and Malnersic, 1980
		Sava R. after Ljubljama Yugoslavia	whiting *Leuciscuso cephalus*	trace	Jan and Malnersic, 1980
Trichloro-benzene	1,2,3-TCB	Kokra R., Yugoslavia	brown trout *Salmo trutta*	12 µg/kg	Jan and Malnersic, 1980
		Drava R. after Maribor Yugoslavia	nace *Chondrostoma nasus*	48 µg/kg	Jan and Malnersic, 1980
		Mura R. Basin, Slovenia Yugoslavia	nace *Chondrostoma nasus*	8 µg/kg	Jan and Malnersic, 1980
		Sava R. after Ljubljana	nace *Chondrostoma nasus*	15 µg/kg	Jan and Malnersic, 1980
		Sava R. after Ljubljana	nace *Chondrostoma nasus*	10 µg/kg	Jan and Malnersic, 1980

DETAILS OF OCCURRENCE OF CHLORINATED BENZENES IN AQUATIC BIOTA: OUTSIDE NORTH AMERICA

COMPOUND	ISOMER	LOCATION	SPECIES,	CONCENTRATION	REFERENCE
Trichloro-benzene	1,2,4-TCB	Kokra R., Yugoslavia	brown trout *Salmo trutta*	1 µg/kg	Jan and Malnersic, 1980
		Drava R. after Maribor Yugoslavia	nace *Chondrostoma nasus*	5 µg/kg	Jan and Malnersic, 1980
		Mura R. Basin, Slovenia Yugoslavia	nace *Chondrostoma nasus*	2 µg/kg	Jan and Malnersic, 1980
		Sava R. after Ljubljama, Yugoslavia	nace *Chondrostoma nasus*	3 µg/kg	Jan and Malnersic, 1980
		Sava R. after Ljubljama Yugoslavia	whiting *Leuciscus cephalus*	15 µg/kg	Jan and Malnersic, 1980
Trichloro-benzene	1,3,5-TCB	Kokra R.,	brown trout *Salmo trutta*	1 µg/kg	Jan and Malnersic, 1980
		Drava R. after Maribor Yugoslavia	nace *Chondrostoma nasus*	5 µg/kg	Jan and Malnersic, 1980
		Mara R. Basin, Slovenia Yugoslavia	nace *Chondrostoma nasus*	1 µg/kg	Jan and Malnersic, 1980

DETAILS OF OCCURRENCE OF CHLORINATED BENZENES IN AQUATIC BIOTA: OUTSIDE NORTH AMERICA

COMPOUND	ISOMER	LOCATION	SPECIES'	CONCENTRATION	REFERENCE
Trichloro-benzene	1,3,5-TCB	Sava R. after Ljubljama, Yugoslavia	nace *Chondrostoma nasus*	5 µg/kg	Jan and Malnersic, 1980
		Sava R. after Ljubljama Yugoslavia	whiting *Leuciscus cephalus*	3 µg/kg	Jan and Malnersic, 1980
Tetrachloro-benzene	1,2,3,4-TeCB	Kokra R., Yugoslavia	brown trout *Salmo trutta*	trace	Jan and Malnersic, 1980
		Drava R. after Maribor Yugoslavia	nace *Chondrostoma nasus*	2 µg/kg	Jan and Malnersic, 1980
		Mura R. Basin, Slovenia, Yugoslavia	nace *Chondrostoma nasus*	5 µg/kg	Jan and Malnersic, 1980
		Sava R. after Ljubljama, Yugoslavia	nace *Chondrostoma nasus*	3 µg/kg	Jan and Malnersic, 1980
		Sava R. after Ljubljama, Yugoslavia	whiting *Leuciscus cephalus*	2 µg/kg	Jan and Malnersic, 1980

DETAILS OF OCCURRENCE OF CHLORINATED BENZENES IN AQUATIC BIOTA: OUTSIDE NORTH AMERICA

COMPOUND	ISOMER	LOCATION	SPECIES	CONCENTRATION	REFERENCE
Tetrachloro-benzene	1,2,4,5- and/or 1,2,3,5-TeCB	Kokra R., Yugoslavia	brown trout *Salmo trutta*	2 µg/kg	Jan and Malnersic, 1980
		Drava R. after Maribor Yugoslavia	nace *Chondrostoma nasus*	12 µg/kg	Jan and Malnersic, 1980
		Mura R. Basin Slovenia Yugoslavia	nace *Chondrostoma nasus*	55 µg/kg	Jan and Malnersic, 1980
Tetrachloro-benzene		Sava R. after Ljubljama Yugoslavia	nace *Chondrostoma nasus*	22 µg/kg	Jan and Malnersic, 1980
		Sava R. after Ljubljama Yugoslavia	whiting *Leuciscus cephalus*	15 µg/kg	Jan and Malnersic, 1980

DETAILS OF OCCURRENCE OF CHLORINATED BENZENES IN AQUATIC BIOTA: OUTSIDE NORTH AMERICA

COMPOUND	ISOMER	LOCATION	SPECIES'	CONCENTRATION	REFERENCE
Pentachloro-benzene	QCB	Kokra R., Yugoslavia	brown trout *Salmo trutta*	6 µg/kg	Jan and Malnersic, 1980
		Drava R. after Maribor Yugoslavia	nace *Chondrostoma nasus*	75 µg/kg	Jan and Malnersic, 1980
		Mura R. Basin, Slovenia Yugoslavia	nace *Chondrostoma nasus*	10 µg/kg	Jan and Malnersic, 1980
		Sava R. after Ljubljama Yugoslavia	nace *Chondrostoma nasus*	20 µg/kg	Jan and Malnersic, 1980
		Sava R. after Ljubljama Yugoslavia	whiting *Leuciscus cephalus*	90 µg/kg	Jan and Malnersic, 1980
Hexachloro-benzene	HCB	Kokra R., Yugoslavia	brown trout *Salmo trutta*	50 µg/kg	Jan and Malnersic, 1980
		Drava R. after Maribor Yugoslavia	nace *Chondrostoma nasus*	160 µg/kg	Jan and Malnersic, 1980
		Mura R. Basin, Slovenia Yugoslavia	nace *Chondrostoma nasus*	80 µg/kg	Jan and Malnersic, 1980

DETAILS OF OCCURRENCE OF CHLORINATED BENZENES IN AQUATIC BIOTA: OUTSIDE NORTH AMERICA

COMPOUND	ISOMER	LOCATION	SPECIES,	CONCENTRATION	REFERENCE
Hexachloro-benzene		Sava R. after Ljubljama Yugoslavia	nace *Chondrostoma nasus*	80 µg/kg	Jan and Malnersic, 1980
		Sava R. after Ljubljama Yugoslavia	nace *Chondrostoma nasus*	220 µg/kg	Jan and Malnersic, 1980
		West Germany	various species of fish	349 µg/kg	Brunn and Manz, 1982

Notes: * wet weight
** high concentration attributed to runoff from an industrial chemical storage area
*** mean
ND - not detected
DL - detection limit
NQ - not quantified
Sig - level of significance
BQT - below quantification threshold

TABLE 6

SUMMARY OF OCCURRENCE OF

CHLORINATED BENZENES IN AQUATIC AND TERRESTRIAL BIRDS

COMPOUND	ISOMER	GREAT LAKES	MARITIMES
Trichlorobenzene	1,2,3-TCB		ND-3.3 µg/kg (DL=0.5 µg/kg)
	1,2,4-TCB	ND-27 µg/kg (DL=10 µg/kg)	ND-37 µg/kg (DL=1.0 µg/kg)
	1,3,5-TCB		ND (DL=0.2 µg/kg)
Tetrachlorobenzene	1,2,3,4-TeCB	ND-43 (DL=10 µg/kg)	ND-8.5 µg/kg (DL=0.1 µg/kg)
	1,2,4,5-TeCB	ND-30 ± 0.17 µg/kg (DL=15 µg/kg)	
	1,2,3,5-TeCB +1,2,4,5-TeCB		ND-11 µg/kg (DL=0.2 µg/kg)
Pentachlorobenzene	QCB	14(±11)-30(±24) µg/kg	ND-5.5 µg/kg (DL=0.1 µg/kg)
Hexachlorobenzene	HCB	0.09-600 µg/kg (maximum of 14700 µg/kg as dry weight)	0.7-62 µg/kg

Notes: ND, not detected
DL, detection limit

DETAILS OF OCCURRENCE OF CHLORINATED BENZENES IN AQUATIC AND TERRESTRIAL BIRDS

COMPOUND	ISOMER	LOCATION	SPECIES	CONCENTRATION	REFERENCE
Trichlorobenzene	1,2,3 -TCB	Maritime Provinces	herring gull eggs	ND-3.3 µg/kg* (DL=0.5 µg/kg)	Matheson et al, 1980
		Maritime Provinces	double-crested cormorant eggs	ND-1.0 µg/kg* (DL=0.5 µg/kg)	Matheson et al, 1980
	1,2,4 -TCB	Great Lakes	herring gull eggs	ND-27 µg/kg	Hallett et al, 1982
		Maritime Provinces	herring gull eggs	ND-37 µg/kg* (DL=1.0 µg/kg)	Matheson et al, 1980
		Maritime Provinces	double-crested cormorant eggs	ND-4.0 µg/kg* (DL=1.0 µg/kg)	Matheson et al, 1980
	1,3,5 -TCB	Maritime Provinces	herring gull eggs	ND* (DL=0.2 µg/kg)	Matheson et al, 1980
		Maritime Provinces	double-crested cormorant eggs	ND* (DL=0.2 µg/kg)	Matheson et al, 1980
Tetrachlorobenzene	1,2,3,4 -TeCB	Great Lakes	herring gull eggs	ND-43 µg/kg	Hallett et al, 1982
		Maritime Provinces	herring gull eggs	ND-11 µg/kg (DL=0.1 µg/kg)	Matheson et al, 1980
		Maritime Provinces	double-crested cormorant eggs	ND-2.6 µg/kg (DL=0.1 µg/kg)	Matheson et al, 1980
	1,2,4,5 -TeCB	Great Lakes	herring gull eggs	ND-30 (± 0.17) µg/kg	Hallett et al, 1982
	1,2,3,5 plus 1,2,4, 5-TeCB	Maritime Provinces	herring gull eggs	ND-11 µg/kg (DL=0.2 µg/kg)	Matheson et al, 1980
		Maritime Provinces	double-crested cormorant eggs	ND-6.4 µg/kg (DL=0.2 µg/kg)	Matheson et al, 1980

DETAILS OF OCCURRENCE OF CHLORINATED BENZENES IN AQUATIC AND TERRESTRIAL BIRDS

COMPOUND	ISOMER	LOCATION	SPECIES	CONCENTRATION	REFERENCE
Pentachlorobenzene	QCB	Great Lakes	herring gull eggs	14(±11)-30(±24) µg/kg	Hallett et al, 1982
		Maritime Provinces	herring gull eggs	ND-4.8 µg/kg (DL=0.1 µg/kg)	Matheson et al.,1980
		Maritime Provinces	double-crested cormorant eggs	ND-5.5 µg/kg (DL=0.1 µg/kg)	Matheson et al.,1980
Hexachlorobenzene	HCB	Delaware R.	immature ruddy ducks	>60-240 µg/kg	White and Kaiser, 1974
		United States	bald eagles	110-500 µg/kg	Cromartie et al., 1975
		Lake Ont. islands	common tern (Sterna sp.)	1350-14700µg/kg**	Gilbertson and Reynolds, 1972
		Great Lakes	herring gull eggs	90(±50)-350(±120) µg/kg	Matheson et al.,1980
		Lake Ontario	herring gull eggs	500 µg/kg	Norstrom et al., 1978
		Maritime Provinces	herring gull eggs	1.0-62 µg/kg (DL=0.1 µg/kg)	Matheson et al.,1980
		Maritime Provinces	double-crested cormorant eggs	0.7-46 µg/kg (DL=0.1 µg/kg)	Matheson et al.,1980
			great skua eggs (Catharacta skua)	600 µg/kg	Furness and Hutton, 1979

TABLE 6 (Page 4)

DETAILS OF OCCURRENCE OF CHLORINATED BENZENES IN AQUATIC AND TERRESTRIAL BIRDS

COMPOUND	ISOMER	LOCATION	SPECIES	CONCENTRATION	REFERENCE
Hexachlorobenzene	HCB	Bay of Fundy	double crested cormorant eggs	15-17 µg/kg	Zitko, 1976
		Lake Erie and Detroit River	herring gull eggs	3500-3900 µg/kg	Mineau 1982
		Southeastern Washington State	birds, raptors	ND-52000 µg/kg	Bechard, 1981
		Bathurst New Brunswick	common tern eggs	28 µg/kg	Holden, 1973
		Hamilton Ontario	common tern eggs	2300 µg/kg	Holden, 1973

ND - not detectable
* - fresh weight
** - dry weight

TABLE 7

OCCURRENCE OF CHLORINATED BENZENES IN THE NIAGARA RIVER

Compound	Isomer	Raw Water	Drinking Water		Sediment		Fish* Spottail Shiners (Young-of-the-Year)
			Raw	Treated	Suspended	Bottom	
monochlorobenzene	-			ND-0.021 µg/L			
dichlorobenzene	DCB		ND-<0.01 µg/L	ND-<0.001 µg/L			
trichlorobenzene	1,2,3						ND-13 µg/kg (DL=2 µg/kg)
	1,2,4				trace		ND-13 µg/kg (DL=2 µg/kg)
	1,3,5						
tetrachlorobenzene	1,2,3,4				19-145 µg/kg		ND-220 µg/kg (DL=2 µg/kg)
	1,2,3,5						ND-43 µg/kg (DL=2 µg/kg)
	1,2,4,5				3-10 µg/kg		ND (DL=2 µg/kg)
pentechlorobenzene	-				23-115 µg/kg	ND-250 µg/kg	ND-18 µg/kg
hexachlorobenzene	-	<0.001 µg/L	ND-0.005 µg/L	ND	17-160 (±535) µg/kg		

Note: * Wet weight basis

Data is summarized from several tables from Environment Canada/MOE, 1981

In a bioaccumulation study with freshwater clams (Elliptio complanatus), hexachlorobenzene was reported at levels from not detectable (detection limit of 1 µg/kg) to 4 µg/kg with the bulk of the estimates being reported as trace levels only.

TABLE 8

HALF-LIVES OF CHLORINATED BENZENES IN WATER, SEDIMENT AND BIOTA

Compound	Isomer	Half-Life in Water (Hrs) Observed	Half-Life in Sediment (Yrs) Observed	Half-Life in Biota (Hrs) Calculated[7]	Observed
Monochlorobenzene	-	9[1]	-	9.2	-
Dichlorobenzene	1,2	<9[1]	-	13.1	-
	1,3	-	-	13.1	-
	1,4	<9[1]	-	15.8	~50[8], ~24[9], 26[10], 14[11]
Trichlorobenzene	1,2,3	-	-	27.8	~150[8]
	1,2,4	7.2[1], 0.75[2]	-	23.2	29[11], <168[12]
	1,3,5	-	-	29.2	~150[8]
Tetrachlorobenzene	1,2,3,4	-	-	-	-
	1,2,3,5	-	-	-	~250[8]
	1,2,4,5	-	-	47.2	49[11], 15[13]
Pentachlorobenzene	-	-	0.74[5]	86	~650[8]
Hexachlorobenzene	-	41[3], 31[3]	4.2[5], ∞[6]	245	~950[8], 291[10], 7 months - several years[14], 240[15], 100-600[16]

Notes:

1 Calculated half-life by Callahan et al., 1979 using observed data reported by Garrison and Hill, 1972 in unagitated distilled water.

2 Calculated half-life by Ware and West, 1977 using data of Simmons et al., 1976 assuming 10 cm deep water column.

3 Laboratory result of I. Scheunerte (unpublished, cited in Schauerte et al, 1982), 20°C and 1 metre water depth.

4 Experimental pond (0.85 m depth) result of Schauerte et al., 1982.

5 Half-life in soil, average of 2 tests by Beck and Hansen, 1974.

6 No degradation observed in simulated pasture study from 2-4 cm depth layer after 19 months. Half-life in surface layer less than 2 weeks (Beall, 1975).

7 Calculated half-life values using the method of Neely, 1979 as cited in Munro et al., 1983. Calculation were based on molecular weight, vapour pressures and calculated solubilities shown in Table 2.

8 Half-life estimates in lipid of guppies (Poecilia reticulata) interpolated from graphs of depuration response in CB-free water. Lipid content of fish 5.4%. (Konemann and van Leeuwen, 1980).

9 Half-life of depuration in the tissues of bluegill sunfish (Lepomis macrochirus) Barrows et al., 1980 as cited in Calamari et al., 1982.

10 Half-life of depuration in muscle of rainbow trout, Salmo gairdneri (Neely et al. 1974).

11 Half-life of depuration in lipids of flagfish (Jordanella floridae). Lipid content of fish 8.5%. (ATRG, 1982 a,b,c).

12 Half-life of depuration from whole body of bluegill sunfish, Lepomis macrochirus (Macek et al., 1979).

13 Half-life of depuration in lipids of brook trout (Salvelinus fontinalis). Lipid content of fish 2.62% (ATRG, 1982c).

14 Half-life of depuration in the tissues of subadult rainbow trout, Salmo gairdneri taking into account increasing body weight (Mimi and Cho, 1981).

15 Half-life of depuration from whole body of green sunfish, Lepomis cyanellus (Sanborn et al., 1977).

16 Range of half-lives from whole body extracts of crayfish (Procambarus clarki), sailfin mollies (Poecilia latipinna), green algae (Oedogonium cardiacum) and largemouth bass (Micropterus salmoides) from Laseter et al., 1976.

TABLE 9

COMPARISON OF CALCULATED PARTITION COEFFICIENTS
WITH SELECTED FIELD AND LABORATORY OBSERVATIONS

COMPOUND/ISOMER	SEDIMENT PARTITION COEFFICIENT (K_{OC}) log K_{OC}[1]	BIOCONCENTRATION FACTOR (K_B) log K_B[2]	LAKE ONTARIO[3] SEDIMENT log K_{OC}	LAKE ONTARIO[3] FISH log K_B	LAKE HURON[3] SEDIMENT log K_{OC}	LAKE HURON[3] FISH log K_B	NIAGARA RIVER[4] SEDIMENT log K_{OC}	NIAGARA RIVER[4] FISH log K_B
Monochlorobenzene								
MCB	2.40	1.29 (2.81-3.62)	-	-	-	-	-	-
Dichlorobenzene								
1,2	3.08	1.97 (1.95-2.74)	3.34	2.30	ND	ND	-	-
1,3	3.23	2.02 (1.82-2.87)	ND	ND	ND	ND	-	-
1,4	3.01	1.90 (1.78-2.86)	3.32	1.65	0.60	2.40	-	-
Trichlorobenzene								
1,2,3	3.67	2.53 (2.85-3.51)	4.85	3.77	ND	ND	-	-
1,2,4	3.83	2.72 (2.26-3.37)	5.19	3.77	1.48	3.70	ND	ND
1,3,5	3.73	2.62 (2.88-3.61)	5.77	4.60	ND	ND	-	-
Tetrachlorobenzene								
1,2,3,4	4.52	3.41 (3.72-4.08)	5.52	4.90	1.30	4.60	4.44(ss)[5]	3.12
1,2,3,5	4.54	3.43 (3.26-3.59)	ND	ND	ND	ND	ND	ND
1,2,4,5	4.46	3.35 (2.74-4.11)	5.71	4.54	ND	ND	3.54(ss)	ND
Pentachlorobenzene								
QCB	5.10	3.99 (3.53-4.30)	5.20	4.77	1.40	4.70	4.99(ss)	3.81
Hexachlorobenzene								
HCB	5.77	4.66 (1.20-4.30)	6.21	6.18	1.70	5.80	5.35	4.65

Notes: [1] Calculated by method of Karickoff et al., 1979 (K_{OC}= 0.63 K_{OW}) from data presented in Table 2, notes 2 and 6, for the organic carbon portion of the sediment only, other material (eg. sand) will alter the measured partition coefficient for any particular sediment.

[2] First value reported is calculated by the method of Mackay, 1982 (K_B= 0.048 K_{OW}) using calculated K_{OW} from Table 2, notes 2 and 6. Values within parentheses are the range of log K_B values (whole body) determined in laboratory uptake studies which are summarized in Table 9. Underscored values are for rainbow trout after 105 days exposure (Oliver and Niimi, 1983).

Note: HCB did not reach steady state during the duration of this study.

[3] Oliver and Nicol, 1982 - Lake Trout, whole body BCF, fish with lipid content of 21%.

[4] Environment Canada/MOE, 1981 - Spottail shiners. Water concentrations from Oliver and Nicol, 1982.

[5] Sediments are bottom sediments unless noted ss for suspended sediments.

[6] ND not detected in one or more of water, sediment or fish thereby preventing calculation of a partition coefficient.

TABLE 10

SUMMARY OF RESULTS OF BIOCONCENTRATION EXPERIMENTS

REFERENCE	ISOMER	SPECIES	TISSUE	EXPOSURE DURATION (days)	EXPOSURE CONCENTRATION (ug/L)	RESIDUE CONCENTRATION (ug/kg)	BIOCONCENTRATION FACTOR BCF	log K_B	COMMENTS
Aquatic Toxicity Research Group, 1982a	1,4-DCB	Flagfish (Jordanella floridae)	whole body	28 uptake 14 depuration	2.68±0.23 3.28±0.27*		296 429*	2.47 2.63*	Flow-through system. BCF based on uptake rate over clearance rate (K_1/K_2). Acetone carrier used.
Aquatic Toxicity Research Group, 1982b	1,2,4-TCB	Flagfish (Jordanella floridae)	whole body	"	3.8 ±0.23*		2026*	3.31*	Flow-through system. BCF based on uptake rate over clearance rate (K_1/K_2). Acetone carrier used.
Aquatic Toxicity Research Group, 1982c	1,2,4,5-TeCB	Flagfish (Jordanella floridae)	whole body	"	3.0 ±0.29 3.87±0.29*		4050 7048*	3.61 3.85*	Flow-through system. BCF based on uptake rate over clearance rate (K_1/K_2). Acetone carrier used.
					*signifies triple CB mixture employed in exposure water				
Aquatic Toxicity Research Group, 1982c	1,2,4,5-TeCB	Brook trout (Salvelinus fontinalis)	whole body	"	4.58±0.18		555	2.74	Flow-through system. BCF based on uptake rate over clearance rate (K_1/K_2). Acetone carrier used.
Calamari et al., 1982	1,4-DCB	Rainbow trout (Salmo gairdneri)	whole body	7 7 7	3 15 73		112 40 85	2.05 1.60 1.93	Continuous flow system.
	1,4-DCB	Rainbow trout (Salmo gairdneri)	whole body	15 23 30 60	3 3 3 3		~400 ~1400 ~300 ~100	2.60 3.15 2.48 2.00	Continuous flow system. BCF at different development stages from egg to alevin over 60 days. Highest BCF in hatched embryos.
Isensee et al., 1976	HCB	Alga (Oedogonium cardiacum)	whole body	33	0.03/0.22/1.72	19-1561	611-910	2.79-2.96	Model Ecosystem. [^{14}C] HCB treated soil or sand placed in bottom of glass aquaria. Static system. Exposure concentrations measured on day 31.
	HCB	Snail (Helisoma sp.)	whole body	33		52-2805	1360-1630	3.13-3.32	
	HCB	Cladoceran (Daphnia magna)	whole body	30		32-1612	940-1030	2.97-3.01	
	HCB	Channel catfish (Ictalurus punctatus)	whole body	8		191-27262	6160-15840	3.79-4.20	
	HCB	Mosquito fish (Gambusia affinis)	whole body	3		39-3519	1260-2040	3.10-3.31	

TABLE 10 (Page 7)

RESULTS OF BIOCONCENTRATION EXPERIMENTS

REFERENCE	ISOMER	SPECIES	TISSUE	EXPOSURE DURATION (days)	EXPOSURE CONCENTRATION (ug/L)	RESIDUE CONCENTRATION (ug/kg)	BIOCONCENTRATION FACTOR BCF	BIOCONCENTRATION FACTOR log K_B	COMMENTS
Konemann and van Leeuwen, 1980	1,4-DCB	Guppy (Poecilia reticulata)	lipid	19	116		1800	3.26	Acetone solvent in a continuous flow system. Exposure concentration measured on day 2. Lipid content 5.42.
	1,2,3-TCB			19	48		13000	4.11	
	1,3,5-TCB			19	43		14000	4.15	
	1,2,3,5-TeCB			19	12		72000	4.86	
	QCB			19	1.2		260000	5.42	
	HCB			19	0.3		290000	5.46	
Laseter et al., 1976	HCB	Crayfish (Procambarus clarki)	whole body	10	31.7 (avg. of 2 tests)	4460 males 5140 females	141 162	2.15 2.21	Flow-through system. Acetone carrier. Steady state not reached.
	HCB	Sailfin mollies (Poecilia latipinna)	whole body	10	62.2	149100	2397	3.38	Flow-through system. Acetone carrier. Steady state not reached.
			whole body	10	7.9	17700	2241	3.35	
	HCB	Largemouth bass (Micropterus salmoides)	whole body	15	9	163920	18214	4.26	Flow-through system. Acetone carrier. Steady state not reached.
			whole body	15	10	325130	32513	4.51	
			whole body	15	2	88880	44440	4.65	
			whole body	15	2	67650	33824	4.53	
	HCB	Green alga (Oedogonium cardiacum)	whole body	7	11.5	7161	623	2.79	Flow-through system. Acetone carrier. Steady state not reached.
	HCB	Bluegill (Lepomis macrochirus)	whole body	7	2.7	3578	1325	3.12	Flow-through system. Acetone carrier.
			whole body	7	HCB-free water with dietary exposure to mollies @ 16ug/g	994			
Lu and Metcalf, 1975	MCB	Algae (Oedogonium cardiacum)	whole body	2	1.01	4205.5	4185	3.62	Static system. [^{14}C] MCB applied. Volatilization losses measured.
	MCB	Snails (Physa sp.)	whole body	2	1.01	1305	1313	3.12	
	MCB	Gladoceran (Daphnia magna)	whole body	2	1.01	2817.3	2789	3.45	
	MCB	Mosquito larvae (Culex quinquifasciatus)	whole body	2	1.01	1327	1292	3.11	
	MCB	Mosquito fish (Gambusia affinis)	whole body	1	1.01	652.1	645	2.81	

TABLE 10 (Page 3)

RESULTS OF BIOCONCENTRATION EXPERIMENTS

REFERENCE	ISOMER	SPECIES	TISSUE	EXPOSURE DURATION (days)	EXPOSURE CONCENTRATION (ug/L)	RESIDUE CONCENTRATION (ug/kg)	BIOCONCENTRATION FACTOR BCF	BIOCONCENTRATION FACTOR log K_B	COMMENTS
Lu and Metcalf, 1975	HCB	Alga (Oedogonium cardiacum)	whole body	2	9.34	37071.8	3969	3.60	Static system. [^{14}C] HCB applied. Volatilization losses measured.
	HCB	Snail (Physa sp.)	whole body	2	9.34	24490.1	2672	3.42	
	HCB	Cladoceran (Daphnia magna)	whole body	2	9.34	10553.7	1129	3.05	
	HCB	Mosquito larvae (Culex quinquifasciatus)	whole body	2	9.34	24960.9	2622	3.42	
	HCB	Mosquito fish (Gambusia affinis)	whole body	2	9.34	10894.1	1166	3.07	
Macek et al., 1979	1,2,4-TCB	Cladoceran (Daphnia magna)	whole body	(steady state reached between 4-8 hours)	3.1±0.4	440±160	142	2.15	Flow-through system. Only total [^{14}C] measured therefore metabolic products not distinguished.
		Bluegill (Lepomis macrochirus)	whole body	28 (steady state within 3 days)	2.9±1.1	530±250	182	2.26	
			whole body	28 (steady state within 3-7 days)	2.9 water exposure plus 440 ug/kg dietary source (daphnia)	570±500	196	2.29	
			whole body	28 (steady state within 14 days)	Dietary exposure to daphnia alone @ 440 ug/kg	30±10			Dietary accumulation approximately 6% of total body burden observed for aqueous and dietary, or aqueous exposure alone.
Melancon et al. (undated abstract)	1,2,4-TCB	Rainbow trout (Salmo gairdneri)	muscle	8 hour 35 4 4	12 20 1 10		)approx.100))approx.400)	2.00 2.60	Static system. Continuous flow system.

TABLE 10 (Page 4)

RESULTS OF BIOCONCENTRATION EXPERIMENTS

REFERENCE	ISOMER	SPECIES	TISSUE	EXPOSURE DURATION (days)	EXPOSURE CONCENTRATION (ug/L)	RESIDUE CONCENTRATION (ug/kg)	BIOCONCENTRATION FACTOR BCF	log K_B	COMMENTS
Metcalf et al., 1973	HCB	Alga (Oedogonium cardiacum)	whole body	33	2.98		522	2.71	Model ecosystem with sloping terrestrial-aquatic interface.
	HCB	Cladoceran (Daphnia magna)	whole body	33	2.98		200	2.30	Salt marsh caterpillars and their feces contaminated the water phase after eating sorghum leaves from radiolabeled HCB seedlings grown on the terrestrial portion
	HCB	Snail (Physa sp.)	whole body	33	2.98		1248	3.10	
	HCB	Mosquito larvae (Culex quinquefasciatus)	whole body	33	2.98		143	2.15	
	HCB	Mosquito fish (Gambusia affinis)	whole body	33	2.98		287	2.46	
Neely et al., 1974	1,4-DCB	Rainbow trout (Salmo gairdneri)	muscle	4			215	2.33	BCF based on uptake rate over clearance rate (K_1/K_2)
Neely et al., 1974	HCB	Rainbow trout (Salmo gairdneri)	muscle	4			7880	3.90	
Oliver and Niimi, 1983	1,2-DCB	Rainbow trout (Salmo gairdneri)	whole body	119	.047 ± .017	11 -14	270	2.43	Continuous flow system, chlorinated benzene mixture in methanol (low exposure)
	1,3-DCB		whole body	119	.028 ± .005	11 -14	420	2.62	
	1,4-DCB		whole body	119	.028 ± .005	7.9-14	370	2.56	
	1,3,5-TCB		whole body	119	.0023 ± .0004	3.7-4.6	1800	3.26	
	1,2,4-TCB		whole body	119	.0032 ± .0002	3.7-8.8	1300	3.11	
	1,2,3-TCB		whole body	119	.0043 ± .0003	4.4-6.6	1200	3.08	
	1,2,4,5-TecB		whole body	119	.0010 ± .0005	2.6-6.9	5300	3.72	
	1,2,3,4-TecB		whole body	119	.0014 ± .0003	2.8-7.7	5200	3.72	
	QCB		whole body	119	.00034 ± .0001	1.2-5.2	13000	4.11	
	HCB		whole body	119	.00032 ± .0003	2.9-6.0	12000*	4.08*	
Oliver and Niimi, 1983	1,2-DCB	Rainbow trout (Salmo gairdneri)	whole body	105	.94 ± .16	360 -670	560	2.74	(high exposure)
	1,3-DCB		whole body	105	.69 ± .15	330 -640	740	2.87	
	1,4-DCB		whole body	105	.67 ± .18	300 -540	720	2.86	
	1,3,5-TCB		whole body	105	.045 ± .02	81 -200	4100	3.61	
	1,2,4-TCB		whole body	105	.052 ± .021	82 -190	3200	3.51	
	1,2,3-TCB		whole body	105	.072 ± .025	96 -210	2600	3.42	
	1,2,4,5-TecB		whole body	105	.021 ± .013	76 -320	13000	4.11	
	1,2,3,4-TeCB		whole body	105	.026 ± .015	79 -350	12000	4.08	
	QCB		whole body	105	.0093 ± .0076	34 -220	20000	4.30	
	HCB		whole body	105	.008 ± .006	13 -160	20000*	4.30*	*HCB did not appear to equilibrate during duration of study

TABLE 10 (Page 5)

RESULTS OF BIOCONCENTRATION EXPERIMENTS

REFERENCE	ISOMER	SPECIES	TISSUE	EXPOSURE DURATION (days)	EXPOSURE CONCENTRATION (ug/L)	RESIDUE CONCENTRATION (ug/kg)	BIOCONCENTRATION FACTOR BCF	BIOCONCENTRATION FACTOR log K_B	COMMENTS
U.S. EPA, 1978	1,2-DCB	Bluegill (Lepomis macrochirus)	whole body	14			89	1.95	
	1,3-DCB		whole body	14			66	1.82	
	1,4-DCB		whole body	14			60	1.78	
U.S. EPA, 1978	1,2,4-TCB	Bluegill (Lepomis macrochirus)	whole body	28			182	2.26	
	1,2,3,5-TeCB		whole body	28			1800	3.26	
	QCB		whole body	28			3400	3.53	
U.S. EPA, 1980a	HCB	Bluegill (Lepomis macrochirus)	whole body	30			22000	4.34	
Veith et al., 1979a	HCB	Fathead minnow (Pimephales promelas)	whole body	32	2.6		16200	4.21	Continuous flow system. Acetone carrier.
		Green sunfish (Lepomis cyanellus)	whole body	32	2.6		21900	4.34	
		Rainbow trout (Salmo gairdneri)	whole body	32	2.6		5500	3.74	
	1,2,4-TCB	Fathead minnow (Pimephales promelas)	whole body	32	1.6		2100	3.32	
		Green sunfish (Lepomis cyanellus)	whole body	32	1.6		2300	3.37	
		Rainbow trout (Salmo gairdneri)	whole body	32	1.6		890	2.95	
Zitko and Hutzinger, 1976	HCB	Atlantic salmon (Salmo salar)	lipid	2	6.6 @ T = 0 d 0.95@ T = 2 d 0.22 @ T = 4d	955 @ T = 2 d 580 @ T = 4 d	690	2.84	Static flow system employing mixture combining 6 chlorophenols. Lipid content 2.31%. Actual concentrations observed would suggest a BCF higher than the 690 reported.

TABLE 11

ACUTE TOXICITY TO AQUATIC ANIMALS

COMPOUND	ISOMER	SPECIES	EFFECT	EXPOSURE	REFERENCE
MONOCHLOROBENZENE					
Monochlorobenzene	-	Cladoceran *Daphnia magna*	24 hr IC_{50}	4,300 µg/L (S,M)	Calamari et al., 1983
Monochlorobenzene	-	Cladoceran *Daphnia magna*	48 hr EC_{50}	86,000µg/L (S,U)	U.S. EPA, 1978(a); LeBlanc, 1980
Monochlorobenzene	-	Rainbow trout *Salmo gairdneri*	48 hr LC_{50}	4,100 µg/L (S,M)	Calamari et al., 1983
Monochlorobenzene	-	Zebrafish *Brachydamio rerio*	48 hr LC_{50}	10,500 µg/L (S,M)	Calamari et al., 1983
Monochlorobenzene	-	Goldfish *Carassius auratus*	96 hr LC_{50}	51,620 µg/L (S,U)	Pickering and Henderson, 1966
Monochlorobenzene	-	Fathead Minnow *Pimephales promelas*	96 hr LC_{50}	33,930 µg/L (S,U) Hardness 20 mg/L	Pickering and Henderson, 1966
				29,120 µg/L (S,U) Hardness 20 mg/L	
				33,930 µg/L (S,U) Hardness 360 mg/L	
Monochlorobenzene	-	Guppy *Poecilla reticulata*	96 hr LC_{50}	45,530 µg/L (S,U)	Pickering and Henderson, 1966
Monochlorobenzene	-	Bluegill *Lepomis macrochirus*	96 hr LC_{50}	24,000 µg/L (S,U)	Pickering and Henderson, 1966

TABLE 11 (Page 2)

ACUTE TOXICITY TO AQUATIC ANIMALS

COMPOUND	ISOMER	SPECIES	EFFECT	EXPOSURE	REFERENCE
Monochlorobenzene	-	Bluegill *Lepomis macrochirus*	96 hr LC_{50}	15,900 µg/L (S,U)	U.S. EPA, 1978(a)
Monochlorobenzene	-	Rainbow trout *Salmo gairdneri*	96 hr LC_{50}	4,700 µg/L (FT,M)	Dalich et al., 1982
Monochlorobenzene	-	Guppy *Poecilla reticulata*	96 hr LC_{50}	23,000 µg/L (FT,M)	Gingerich and Glen, unpublished
Monochlorobenzene		Bluegill *Lepomis macrochirus*	96 hr LC_{50}	16,000 µg/L (S,U)	Buccafusco et al., 1981
Monochlorobenzene	-	Goldfish (embryo-larval) *Carassius auratus*	8 day LC_{50}	880 µg/L at 50 mg/L Hardness (FT,M)	Birge et al., 1979
Monochlorobenzene	-	Goldfish (embryo-larval) *Carassius auratus*	8 day LC_{50}	1,040 µg/L at 200 mg/L Hardness (FT,M)	Birge et al., 1979
Monochlorobenzene	-	Largemouth bass (embryo-larval) *Micropterus salmoides*	7.5 day LC_{50}	50 µg/L at 50 mg/L Hardness (FT,M)	Birge et al., 1979
Monochlorobenzene	-	Largemouth bass (embryo-larval) *Micropterus salmoides*	7.5 day LC_{50}	60 µg/L at 200 mg/L Hardness (FT,M)	Birge et al., 1979
Monochlorobenzene	-	Rainbow trout (embryo) *Salmo gairdneri*	16 day LC_{50}	100% mortality at 90 µg/L (FT,M)	Birge et al., 1979

TABLE 11 (Page 3)

ACUTE TOXICITY TO AQUATIC ANIMALS

COMPOUND	ISOMER	SPECIES	EFFECT	EXPOSURE	REFERENCE
DICHLOROBENZENE					
Dichlorobenzene	1,2	Cladoceran *Daphnia magna*	24 hr IC_{50}	780 µg/L (S,M)	Calamari et al., 1983
Dichlorobenzene	1,2	Cladoceran *Daphnia magna*	48 hr EC_{50}	2,440 µg/L (S,U)	U.S. EPA, 1978(a)
Dichlorobenzene	1,2	Midge *Tanytarsus dissimilis*	48 hr EC_{50}	11,760 µg/L (S,M)	U.S. EPA, 1978(a); LeBlanc, 1980
Dichlorobenzene	1,2	Rainbow trout *Salmo gairdneri*	48 hr LC_{50}	2,300 µg/L (S,M)	Calamari et al., 1983
Dichlorobenzene	1,2	Zebrafish *Brachydanio rerio*	48 hr LC_{50}	6,800 µg/L (S,M)	Calamari et al., 1983
Dichlorobenzene	1,2	Rainbow trout *Salmo gairdneri*	96 hr LC_{50}	1,580 µg/L (FT,M)	U.S. EPA, 1980(b)
Dichlorobenzene	1,2	Bluegill *Lepomis macrochirus*	96 hr LC_{50}	27,000 µg/L (S,U)	Dawson et al., 1977
Dichlorobenzene	1,2	Bluegill *Lepomis macrochirus*	96 hr LC_{50}	5,590 µg/L (S,U)	U.S. EPA, 1978(b)
Dichlorobenzene	1,2	Bluegill *Lepomus macrochirus*	96 hr LC_{50}	5,600 µg/L (S,U)	Buccafusco et al., 1981
Dichlorobenzene	1,2	Fathead Minnow *Pimephales promelas*	96 hr LC_{50}	57,000 µg/L (S,U)	Curtis and Ward, 1981

TABLE 11 (Page 4)

ACUTE TOXICITY TO AQUATIC ANIMALS

COMPOUND	ISOMER	SPECIES	EFFECT	EXPOSURE	REFERENCE
Dichlorobenzene	1,3	Cladoceran *Daphnia magna*	48 hr EC_{50}	28,100 µg/L (S,U)	U.S. EPA, 1978(a); LeBlanc, 1980
Dichlorobenzene	1,3	Fathead Minnow *Pimephales promelas*	96 hr LC_{50}	7,790 µg/L (FT,M)	U.S. EPA, 1980(b)
Dichlorobenzene	1,3	Bluegill *Lepomis macrochirus*	96 hr LC_{50}	5,020 µg/L (S,U)	U.S. EPA, 1978(a)
Dichlorobenzene	1,3	Bluegill *Lepomis macrochirus*	96 hr LC_{50}	5,000 µg/L (S,U)	Buccafusco et al., 1981
Dichlorobenzene	1,4	Cladoceran *Daphnia magna*	24 hr IC_{50}	1,600 µg/L (S,M)	Calamari et al., 1982; 1983
Dichlorobenzene	1,4	Cladoceran *Daphnia magna*	48 hr EC_{50}	11,000 µg/L (S,U)	U.S. EPA, 1978(a); LeBlanc, 1980
Dichlorobenzene	1,4	Midge *Tanytarsus dissimilis*	48 hr EC_{50}	13,000 µg/L (S,M)	U.S. EPA, 1980(b)

TABLE 11 (Page 5)

ACUTE TOXICITY TO AQUATIC ANIMALS

COMPOUND	ISOMER	SPECIES	EFFECT	EXPOSURE	REFERENCE
Dichlorobenzene	1,4	Rainbow trout *Salmo gairdneri*	48 hr LC_{50}	1,200 µg/L (S,M)	Calamari et al., 1982; 1983
Dichlorobenzene	1,4	Zebrafish *Brachydanio rerio*	48 hr LC_{50}	4,300 µg/L (S,M)	Calamari et al., 1982; 1983
Dichlorobenzene	1,4	Rainbow trout *Salmo gairdneri*	96 hr LC_{50}	1,120 µg/L (FT,M)	U.S. EPA, 1980(b)
Dichlorobenzene	1,4	Fathead minnow *Pimephales promelas*	96 hr LC_{50}	4,000 µg/L (FT,M)	U.S. EPA, 1980(b)
Dichlorobenzene	1,4	Bluegill *Lepomis macrochirus*	96 hr LC_{50}	4,280 µg/L (S,U)	U.S. EPA, 1978(a)
Dichlorobenzene	1,4	Bluegill *Lepomis macrochirus*	96 hr LC_{50}	4,300 µg/L (S,U)	Buccafusco et al., 1981
Dichlorobenzene	1,4	Fathead minnow *Pimephales promelas*	96 hr LC_{50}	30,000 µg/L (S,U)	Curtis and Ward, 1981
Dichlorobenzene	1,4	Flagfish *Jordanella floridae*	96 hr LC_{50}	4,490 µg/L (S,U)	ATRG, unpublished b.
Dichlorobenzene	1,4	Flagfish *Jordanella floridae*	96 hr LC_{50}	2,050 µg/L (FT,M)	ARTG, unpublished b.
Dichlorobenzene	1,4	Rainbow trout *Salmo gairdneri*	14 day LC_{50}	800 µg/L (FT,M)	Calamari et al., 1982

ACUTE TOXICITY TO AQUATIC ANIMALS

COMPOUND	ISOMER	SPECIES	EFFECT	EXPOSURE	REFERENCE
TRICHLOROBENZENE					
Trichlorobenzene	1,2,3	Cladoceran *Daphnia magna*	24 hr IC_{50}	350 µg/L (S,M)	Calamari et al., 1983
Trichlorobenzene	1,2,3	Rainbow trout *Salmo gairdneri*	48 hr LC_{50}	710 µg/L (S,M)	Calamari et al., 1983
Trichlorobenzene	1,2,3	Zebrafish *Brachydanio rerio*	48 hr LC_{50}	3,100 µg/L (S,M)	Calamari et al., 1983
Trichlorobenzene	1,2,4	Cladoceran *Daphnia magna*	24 hr IC_{50}	1,200 µg/L (S,M)	Calamari et al., 1983
Trichlorobenzene	1,2,4	Rainbow trout *Salmo gairdneri*	48 hr LC_{50}	1,950 µg/L (S,M)	Calamari et al., 1983
Trichlorobenzene	1,2,4	Zebrafish *Brachydanio rerio*	48 hr LC_{50}	6,300 µg/L (S,M)	Calamari et al., 1983
Trichlorobenzene	1,2,4	Cladoceran *Daphnia magna*	48 hr EC_{50}	50,200 µg/L (S,U)	U.S. EPA, 1978(a); LeBlanc, 1980
Trichlorobenzene	1,2,4	Rainbow trout *Salmo gairdneri*	96 hr LC_{50}	1,500 µg/L (FT,M)	U.S. EPA, 1980(a)
Trichlorobenzene	1,2,4	Fathead minnow *Pimephales promelas*	96 hr LC_{50}	2,870 µg/L (FT,M)	U.S. EPA, 1980(a)

ACUTE TOXICITY TO AQUATIC ANIMALS

COMPOUND	ISOMER	SPECIES	EFFECT	EXPOSURE	REFERENCE
Trichlorobenzene	1,2,4	Bluegill *Lepomis macrochirus*	96 hr LC_{50}	3,369 µg/L (S,U)	U.S. EPA, 1978(a)
Trichlorobenzene	1,2,4	Bluegill *Lepomis macrochirus*	96 hr LC_{50}	4,300 µg/L (S,U)	Buccafusco et al., 1982
Trichlorobenzene	1,2,4	Flagfish *Jordanella floridae*	96 hr LC_{50}	3,990 µg/L (S,U)	ATRG, unpublished c
Trichlorobenzene	1,2,4	Flagfish *Jordanella floridae*	96 hr LC_{50}	1,220 µg/L (FT,M)	ATRG, unpublished c
Trichlorobenzene	66% 1,2,4 33% 1,2,3	Flagfish *Jordanella floridae*	96 hr LC_{50}	2170-3180 µg/L(FT,M)	Smith et al., 1979
TETRACHLOROBENZENE					
Tetrachlorobenzene	1,2,3,4	Fathead minnow *Pimephales promelas*	96 hr LC_{50}	1,070 µg/L (FT,M)	U.S. EPA, 1980(a)
Tetrachlorobenzene	1,2,3,5	Cladoceran *Daphnia magna*	48 hr EC_{50}	9,710 µg/L (S,U)	U.S. EPA, 1978(a); LeBlanc, 1980
Tetrachlorobenzene	1,2,3,5	Bluegill *Lepomis macrochirus*	96 hr LC_{50}	6,400 µg/L (S,U)	Buccafusco et al., 1981
Tetrachlorobenzene	1,2,3,5	Bluegill *Lepomis macrochirus*	96 hr LC_{50}	6,420 µg/L (S,U)	U.S. EPA, 1978(a)
Tetrachlorobenzene	1,2,4,5	Cladoceran *Daphnia magna*	48 hr EC_{50}	>553,000 µg/L (S,U)	U.S. EPA, 1978(a); LeBlanc, 1980
Tetrachlorobenzene	1,2,4,5	Bluegill *Lepomis macrochirus*	96 hr LC_{50}	1,550 µg/L (S,U)	U.S. EPA, 1978(a)

TABLE 11 (Page 8)

ACUTE TOXICITY TO AQUATIC ANIMALS

COMPOUND	ISOMER	SPECIES	EFFECT	EXPOSURE	REFERENCE
Tetrachlorobenzene	1,2,4,5	Bluegill *Lepomis macrochirus*	96 hr LC_{50}	1,600 µg/L (S,U)	Buccafusco et al., 1981
Tetrachlorobenzene	1,2,4,5	Flagfish *Jordanella floridae*	96 hr LC_{50}	1,570 µg/L (S,U)	ATRG, unpublished d
Tetrachlorobenzene	1,2,4,5	Flagfish *Jordanella floridae*	96 hr LC_{50}	No lethality from dissolved material only at saturation, 500 µg/L (FT,M). Gave LC_{50} of 2000 µg/L based on dissolved and particulate total.	ATRG, unpublished d
PENTACHLOROBENZENE					
Pentachlorobenzene	-	Cladoceran *Daphnia magna*	48 hr EC_{50}	5,280 µg/L (S,U)	U.S. EPA, 1978(a); LeBlanc, 1980
Pentachlorobenzene	-	Bluegill *Lepomis macrochirus*	96 hr LC_{50}	250 µg/L (S,U)	U.S. EPA, 1978(a)
Pentachlorobenzene	-	Bluegill *Lepomis macrochirus*	96 hr LC_{50}	250 µg/L (S,U)	Buccafusco et al., 1981
Pentachlorobenzene	-	Rainbow trout *Salmo gairdneri*	12 day LC_{50}	258 µg/L (FT,M)	U.S. EPA, 1980(a)

ACUTE TOXICITY TO AQUATIC ANIMALS

COMPOUND	ISOMER	SPECIES	EFFECT	EXPOSURE	REFERENCE
Pentachlorobenzene	-	Guppy *Poecilia reticulata*	14 day LC_{50}	<250 µg/L (S)	Konemann, 1979
HEXACHLOROBENZENE					
Hexachlorobenzene	-	Cladoceran *Daphnia magna*	24 hr IC_{50}	no toxicity at saturation 30 µg/L (S,M)	Calamari et al., 1983
Hexachlorobenzene	-	Midge *Tanytarsus disimilis*	48 hr EC_{50} at 57 µg/L	test, no mortality	U.S. EPA, 1980(a)
Hexachlorobenzene	-	Rainbow trout *Salmo gairdneri*	48 hr LC_{50}	no toxicity at saturation 30 µg/L (S,M)	Calamari et al., 1983
Hexachlorobenzene	-	Zebrafish *Brachydanio rerio*	48 hr LC_{50}	no toxicity at saturation 30 µg/L (S,M)	Calamari et al., 1983
Hexachlorobenzene	-	Rainbow trout *Salmo gairdneri*	96 hr LC_{50} at 80 µg/L	test, no mortality	U.S. EPA, 1980(a)
Hexachlorobenzene	-	Fathead minnow *Pimephales promelas*	96 hr LC_{50} at 48 µg/L	test, no mortality	U.S. EPA, 1980(a)
Hexachlorobenzene	-	Bluegill *Lepomis macrochirus*	96 hr LC_{50} at 78 µg/L	test, no mortality	U.S. EPA, 1980(a)

TABLE 11 (Page 10)

ACUTE TOXICITY TO AQUATIC ANIMALS

COMPOUND	ISOMER	SPECIES	EFFECT	EXPOSURE	REFERENCE
Hexachlorobenzene	-	Crayfish *Procambarus clarki*	LC_{50} test at 28-36 µg/L, no mortality attributed to HCB over 20 days		Laska et al., 1976; Laseter et al., 1976

NOTES: FT, flow through test

S, static test

M, measured concentration

U, nominal unmeasured concentration

LC_{50}, exposure concentration lethal to 50% of the exposed organisms

IC_{50}, exposure concentration which immobilizes 50% of the exposed organisms, similar to LC_{50}, used for organisms where death is difficult to determine accurately

EC_{50}, exposure concentration which produces a defined effect in 50% of the exposed organisms, defined effect is usually immobilization or cessation of respiration.

TABLE 12

ACUTE TOXICITY TO AQUATIC ANIMALS: INTRAPERITONEAL INJECTION

Compound	Isomer	Species	Effect / Exposure		Reference
Monochlorobenzene	-	Rainbow trout *Salmo gairdneri*	96 hr/LD_{50}	9.68 m moles/kg	Kaiser et al. in press
Dichlorobenzene	1,2	Rainbow trout *Salmo gairdneri*	96 hr/LD_{50}	7.45 m moles/kg	Kaiser et al. in press
Dichlorobenzene	1,3	Rainbow trout *Salmo gairdneri*	96 hr/LD_{50}	9.8 m moles/kg	Kaiser et al. in press
Dichlorobenzene	1,4	Rainbow trout *Salmo gairdneri*	96 hr/LD_{50}	10.1 m moles/kg	Kaiser et al. in press
Trichlorobenzene	1,2,3	Rainbow trout *Salmo gairdneri*	96 hr/LD_{50}	8.9 m moles/kg	Kaiser et al. in press
Trichlorobenzene	1,2,4	Rainbow trout *Salmo gairdneri*	96 hr/LD_{50}	9.8 m moles/kg	Kaiser et al. in press
Trichlorobenzene	1,3,5	Rainbow trout *Salmo gairdneri*	96 hr/LD_{50}	30.5 m moles/kg	Kaiser et al. in press
Tetrachlorobenzene	1,2,3,4	Rainbow trout *Salmo gairdneri*	96 hr/LD_{50}	5.1 m moles/kg	Kaiser et al. in press
Tetrachlorobenzene	1,2,3,5	Rainbow trout *Salmo gairdneri*	96 hr/LD_{50}	7.7 m moles/kg	Kaiser et al. in press
Tetrachlorobenzene	1,2,4,5	Rainbow trout *Salmo gairdneri*	96 hr/LD_{50}	>18.6 m moles/kg	Kaiser et al. in press
Pentachlorobenzene	-	Rainbow trout *Salmo gairdneri*	96 hr/LD_{50}	5.8 m moles/kg	Kaiser et al. in press
Hexachlorobenzene	-	Rainbow trout *Salmo gairdneri*	96 hr/LD_{50}	> 8.8 m moles/kg	Kaiser et al. in press

NOTES: 1,2,4,5 TeCB value is from an unidentified source cited in Kaiser et al., in press. This isomer was not tested by Kaiser et al. With the experimental design employed it was not possible to inject enough HCB to produce mortality.

The relationship of the above data is $1/\log C = -1.71 + 0.20 \log K_{ow}$ ($r^2 = 0.78$)

where C = LD_{50} in m moles/kg

K_{ow} = calculated octanol-water partition coefficient

TABLE 13

CHRONIC TOXICITY TO AQUATIC ANIMALS

COMPOUND	ISOMER	SPECIES	EFFECT/EXPOSURE	REFERENCE
Monochlorobenzene	-	Rainbow trout *Salmo gairdneri*	Embryo-larval test (4 days post-hatch) 27 day LC_{50} in hard water (100 mg/L) is 110 µg/L (FT,M)	Black et al., 1982
Monochlorobenzene	-	Northwest salamander *Ambystoma gracile*	Embryo-larval test (4 days post hatch) 9.5 day LC_{50} in hard water (100 mg/L) is 1150 µg/L (FT,M)	Black et al., 1982
Monochlorobenzene	-	Leopard frog *Rana pipiens*	Embryo-larval test (4 days post hatch) 90 day LC_{50} in hard water (100 mg/L) is 1200 µg/L (FT,M)	Black et al., 1982
Dichlorobenzene	1,2	Fathead minnow *Pimephales promelas*	Early Life Stage Test Chronic Value of 2000 (limits 1600-2500) µg/L	U.S. EPA, 1978(a)
Dichlorobenzene	1,3	Fathead minnow *Pimephales promelas*	Early Life Stage Test Chronic Value of 1510 (limits 1000-2270) µg/L	U.S. EPA, 1980(b)
Dichlorobenzene	1,4	Fathead minnow *Pimephales promelas*	Early Life Stage Test Chronic Value of 763 (limits 560-1040) µgL	U.S. EPA, 1980(b)

TABLE 13 (Page 2)

CHRONIC TOXICITY TO AQUATIC ANIMALS

COMPOUND	ISOMER	SPECIES	EFFECT/EXPOSURE	REFERENCE
Dichlorobenzene	1,4	Flagfish *Jordanella floridae*	Fry survival and growth over 28 days, Chronic Value >349 µg/L (FT,M)	ATRG, unpublished b
			10 day larval survival Chronic Value 252 to 273 µg/L (FT,M)	ATRG, unpublished b
Trichlorobenzene	1,2,4	Fathead minnow *Pimephales promelas*	Early Life Stage Test Chronic Value of 286 (limits 200-410) µg/L	U.S. EPA, 1978(a)
Trichlorobenzene	1,2,4	Fathead minnow *Pimephales promelas*	Early Life Stage Test Chronic Value of 705 (limits 499-995) µg/L	U.S. EPA, 1980(b)
Trichlorobenzene	1,2,4	Rainbow trout *Salmo gairdneri*	No Observed Effect Level after 85 days (Fry) Range 100-160 µg/L	Hodson, unpublished
Tetrachlorobenzene	1,2,3,4	Fathead minnow *Pimephales promelas*	Early Life Stage Test Chronic Value of 318 (limits 245-412) µg/L	U.S. EPA, 1980(a)
Tetrachlorobenzene	1,2,3,4	Fathead minnow *Pimephales promelas*	Early Life Stage Test Chronic Value of 713 (limits 497-993)	U.S. EPA, 1978(a)

CHRONIC TOXICITY TO AQUATIC ANIMALS

COMPOUND	ISOMER	SPECIES	EFFECT/EXPOSURE	REFERENCE
Tetrachlorobenzene	1,2,4,5	Flagfish *Jordanella floridae*	Fry growth over 28 days, Chronic Value of 85 µg/L	ATRG, unpublished d
Tetrachlorobenzene	1,2,4,5	Brook trout *Salvelinus fontinalis*	Fry survival and growth over 28 days, Chronic Value of <243 µg/L	ATRG, unpublished d
Pentachlorobenzene	-	-	-	-
Hexachlorobenzene	-	Largemouth bass *Micropterus salmoides*	10 and 15 day exposures at 10 and 26 µg/L (FT, M) resulted in no mortality; however, histological damage was noted in the liver, kidney and gall bladder after 10 days exposure to 3.5 µg/L	Laska et al., 1976; Laseter et al., 1976

TABLE 14

EFFECTS OF CHLORINATED BENZENES ON FRESHWATER ALGAE

COMPOUND	ISOMER	SPECIES	EFFECT	NOMINAL EXPOSURE	REFERENCE
Monochlorobenzene	-	Selenastrum capricornutum	Photosynthesis 3 hr EC_{50}	33,000 µg/L	Calamari et al., 1983
Monochlorobenzene	-	Selenastrum capricornutum	Chlorophyll a 96 hr EC_{50}	232,000 µg/L	U.S. EPA, 1978(a)
Monochlorobenzene	-	Selenastrum capricornutum	Cell Numbers 96 hr EC_{50}	224,000 µg/L	U.S. EPA, 1978(a)
Monochlorobenzene	-	Selenastrum capricornutum	Growth 96 hr EC_{50}	12,500 µg/L	Calamari et al., 1983
Monochlorobenzene	-	Selenastrum capricornutum	Fertility 14 day EC_{50}	2,500 µg/L	Calamari et al., 1983
Dichlorobenzene	1,2	Selenastrum capricornutum	Photosynthesis 3 hr EC_{50}	10,000 µg/L	Calamari et al., 1983
Dichlorobenzene	1,2	Selenastrum capricornutum	Chlorophyll a 96 hr EC_{50}	91,600 µg/L	U.S. EPA, 1978(a)
Dichlorobenzene	1,2	Selenastrum capricornutum	Cell Numbers 96 hr EC_{50}	98,000 µg/L	U.S. EPA, 1978(a)
Dichlorobenzene	1,2	Selenastrum capricornutum	Growth 96 hr EC_{50}	2,200 µg/L	Calamari et al., 1983
Dichlorobenzene	1,2	Selenastrum capricornutum	Fertility 14 day EC_{50}	550 µg/L	Callamari et al., 1983

TABLE 14 (Page 2)

EFFECTS OF CHLORINATED BENZENES ON FRESHWATER ALGAE

COMPOUND	ISOMER	SPECIES	EFFECT	NOMINAL EXPOSURE	REFERENCE
Dichlorobenzene	1,3	Selenastrum capricornutum	Chlorophyll a 96 hr EC_{50}	179,000 µg/L	U.S. EPA, 1978(a)
Dichlorobenzene	1,3	Selenastrum capricornutum	Cell Numbers 96 hr EC_{50}	149,000 µg/L	U.S. EPA, 1978(a)
Dichlorobenzene	1,4	Selenastrum capricornutum	Photosynthesis 3 hr EC_{50}	5,200 µg/L	Calamari et al., 1983
Dichlorobenzene	1,4	Selenastrum capricornutum	Chlorophyll a 96 hr EC_{50}	98,100 µg/L	U.S. EPA, 1978(a)
Dichlorobenzene	1,4	Selenastrum capricornutum	Cell Numbers 96 hr EC_{50}	96,700 µg/L	U.S. EPA, 1978(a)
Dichlorobenzene	1,4	Selenastrum capricornutum	Growth 96 hr EC_{50}	1,600 µg/L	Calamari et al., 1983
Dichlorobenzene	1,4	Selenastrum capricornutum	Fertility 14 day EC_{50}	930 µg/L	Calamari et al., 1983
Trichlorobenzene	1,2,3	Selenastrum capricornutum	Photosynthesis 3 hr EC_{50}	200 µg/L	Calamari et al., 1983
Trichlorobenzene	1,2,3	Selenastrum capricornutum	Growth 96 hr EC_{50}	900 µg/L	Calamari et al., 1983
Trichlorobenzene	1,2,3	Selenastrum capricornutum	Fertility 14 day EC_{50}	200 µg/L	Calamari et al., 1983

TABLE 14 (Page 3)

EFFECTS OF CHLORINATED BENZENES ON FRESHWATER ALGAE

COMPOUND	ISOMER	SPECIES	EFFECT	NOMINAL EXPOSURE	REFERENCE
Trichlorobenzene	1,2,4	Selenastrum capricornutum	Photosynthesis 3 hr EC_{50}	3,900 µg/L	Calamari et al., 1983
Trichlorobenzene	1,2,4	Selenastrum capricornutum	Chlorophyll a 96 hr EC_{50}	33,300 µg/L	U.S. EPA, 1978(a)
Trichlorobenzene	1,2,4	Selenastrum capricornutum	Cell Numbers 96 hr EC_{50}	36,700 µg/L	U.S. EPA, 1978(a)
Trichlorobenzene	1,2,4	Selenastrum capricornutum	Growth 96 hr EC_{50}	1,400 µg/L	Calamari et al., 1983
Trichlorobenzene	1,2,4	Selenastrum capricornutum	Fertility 14 day EC_{50}	450 µg/L	Calamari et al., 1983
Tetrachlorobenzene	1,2, 3,5	Selenastrum capricornutum	Chlorophyll a 96 hr EC_{50}	17,200 µg/L	U.S. EPA, 1978(a)
Tetrachlorobenzene	1,2, 3,5	Selenastrum capricornutum	Cell Numbers 96 hr EC_{50}	17,700 µg/L	U.S. EPA, 1978(a)
Tetrachlorobenzene	1,2, 4,5	Selenastrum capricornutum	Chlorophyll a 96 hr EC_{50}	52,900 µg/L	U.S. EPA, 1978(a)
Tetrachlorobenzene	1,2, 4,5	Selenastrum capricornutum	Cell Numbers 96 hr EC_{50}	46,800 µg/L	U.S. EPA, 1978(a)

TABLE 14 (Page 4)

EFFECTS OF CHLORINATED BENZENES ON FRESHWATER ALGAE

COMPOUND	ISOMER	SPECIES	EFFECT	NOMINAL EXPOSURE	REFERENCE
Pentachlorobenzene	-	Selenastrum capricornutum	Chlorophyll a 96 hr EC_{50}	6,780 µg/L	U.S. EPA, 1978(a)
Pentachlorobenzene	-	Selenastrum capricornutum	Cell Numbers 96 hr EC_{50}	6,630 µg/L	U.S. EPA, 1978(a)
Hexachlorobenzene	-	Selenastrum capricornutum	Photosynthesis 3 hr EC_{50}	18 µg/L	Calamari et al., 1983
Hexachlorobenzene	-	Selenastrum capricornutum	Growth 96 hr EC_{50}	<30 µg/L	Calamari et al., 1983
Hexachlorobenzene	-	Selenastrum capricornutum	Fertility 14 day LC_{50}	16 µg/L	Calamari et al., 1983

Note: EC_{50} is the concentration required to produce an effect (reduction) in 50% of the exposed population

Calalmari et al., 1983 report that for all compounds they tested losses from the test water did not exceed 15% after 48 hours, after 48 hours the solution was changed. Closed test chambers were used.

TABLE 15

ODOUR/TASTE THRESHOLDS OF CHLORINATED BENZENES IN WATER

Compound	Isomer	Odour Threshold (µg/L)	Taste Threshold (µg/L)
Monochlorobenzene		100[1]	
Dichlorobenzene	1,2	2[3] - 10[1,2]	0.1[3]
	1,3	-	-
	1,4	0.3[2] - 2[3] - 30[1]	6[3]
Trichlorobenzene	1,2,3	-	
	1,2,4	5[1,2]	
	1,3,5	-	
Tetrachlorobenzene	1,2,3,4	-	
	1,2,3,5	-	
	1,2,4,5	130[1]	
Pentachlorobenzene		-	
Hexachlorobenzene		3000[1]-6[2]	

Notes: [1]-WHO, 1980
[2] Zoeteman et al.,.1980
[3] Varshavskaya, 1967 cited in U.S. EPA, 1980

TABLE 16

SUMMARY OF DATA PRESENTED IN FIGURE 1[1]

COMPOUND	Log Kow	ACUTE TOXICITY 96h LC_{50} μ MOLES/L	CHRONIC TOXICITY μ MOLES/L mean	range	REFERENCE
Monochlorobenzene	2.61	41.8(S)	-	-	Dalich et al., 1982
	2.61	204	-	-	Gingerich and Glen, unpublished
	2.61	0.44[A]	-	-	Birge et al., 1979
Dichlorobenzene					
(1,2)	3.29	10.8	-	-	U.S. EPA, 1980(b)
	3.29	-	13.6	10.9-17	U.S. EPA, 1978(a)
(1,3)	3.34	53	10.3	6.8-15.4	U.S. EPA, 1980(b)
(1,4)	3.22	7.6(S)	-	-	U.S. EPA, 1980(b)
	3.22	27	5.2	1.8-7.1	U.S. EPA, 1980(b)
	3.22	14	>2.4	-	ATRG, unpublished b
	3.22	-	-	1.7-1.9	ATRG, unpublished b
Trichlorobenzene					
(1,2,3)	3.85	3.9(S)[A]	-	-	Calamari et al., 1983
(1,2,3)	4.04	8.3(S)	-	-	U.S. EPA, 1980
	4.04	15.8	3.9	2.8-5.5	U.S. EPA, 1980
	4.04	6.7	-	-	ATRG, unpublished c
	4.04	-	-	0.55-0.88(S)	Hodson et al., unpublished
	4.04	-	1.6	1.1-2.3	U.S. EPA, 1978(a)
Tetrachlorobenzene					
(1,2,3)	4.73	5.0	1.5	1.1-1.9	U.S. EPA, 1980(a)
	4.73	-	3.3	2.3-4.6	U.S. EPA, 1978(a)
(1,2,4,5)	4.67	9.3	0.39	-	ATRG, unpublished d
(1,4)	4.67	-	≤1.1(S)	-	ATRG, unpublished d
Pentachlorobenzene	5.31	1.0(S)[A]	-	-	Several references, see Table 10.
Hexachlorobenzene	5.98	-	0.012[C]	-	Laseter et al., 1976

NOTES:
1. Data are from Tables 10 and 12.
2. (S) indicates data are from a test with a salmonid fish.
3. A, lowest reported concentration producing an effect. It is from an acute test other than of 96 h duration.
4. C, lowest reported concentration producing an effect. It is from a non-standard chronic test.

TABLE 17

ESTIMATED SAFE LEVELS FOR CHLORINATED BENZENES
BASED ON AQUATIC TOXICITY INFORMATION

COMPOUND	ISOMER	MOLECULAR WEIGHT g/MOLE	CHRONIC TOXICITY[1] ESTIMATE µ MOLES/L	SAFETY FACTOR	ESTIMATED SAFE LEVEL µ MOLES/L	µg/L
Monochlorobenzene	-	112.56	25	0.10	2.5	281
Dichlorobenzene						
	1,2	147.01	5.0	0.10	0.50	74
	1,3	147.01	4.3	0.10	0.43	63
	1,4	147.01	7.0	0.10	0.70	103
Trichlorobenzene	1,2,3	181.45	1.7	0.10	0.17	31
	1,2,4	181.45	0.95	0.10	0.095	17
	1,3,5,	181.45	1.2	0.10	0.12	22
Tetrachlorobenzene	1,2,3,4	215.9	0.20	0.10	0.020	4.3
	1,2,3,5	215.9	0.19	0.10	0.019	4.1
	1,2,4,5	215.9	0.25	0.10	0.025	5.4
Pentachlorobenzene	-	250.34	0.060	0.10	0.006	1.5
Hexachlorobenzene	-	284.79	0.015	0.10	0.0015	0.43

NOTE: 1. Values interpolated from chronic toxicity line on Figure 1. Straight portion of line below K_{ow} of about 4.5 used rather than curved portion.

TABLE 18

SUGGESTED PROVINCIAL WATER QUALITY OBJECTIVES FOR CHLORINATED BENZENES

COMPOUND	ISOMER	SUGGESTED CRITERIA µg/L
Monochlorobenzene	-	15
Dichlorobenzene	1,2	2.5
	1,3	2.5
	1,4	4.0
Trichlorobenzene	1,2,3	0.90
	1,2,4	0.50
	1,3,5	0.65
Tetrachlorobenzene	1,2,3,4	0.10
	1,2,3,5	0.10
	1,2,4,5	0.15
Pentachlorobenzene		0.030
Hexachlorobenzene		0.0065

TABLE 19

CRITERIA DEVELOPED BY OTHER JURISDICTIONS

JURISDICTION	REFERENCE	COMPOUND	ISOMER	MAXIMUM WATER CONCENTRATION PROPOSED	BACKGROUND RESEARCH	REFERENCE	CRITERION DEVELOPMENT: SAFETY FACTOR	CRITERION DEVELOPMENT: BIOCONCENTRATION FACTOR	ADI
EPA protection of human health	U.S. EPA, 1980(a)	Monochlorobenzene	MCB	20 ug/L*	dog-NOAEL=27.25 mg/kg/day (3 months)	Knapp, 1971	1000	10.3	1.008 mg/day
					rat-NOAEL=12.5 mg/kg/day	Knapp, 1971			
					rat-NOAEL=14.5 mg/kg/day	Irish, 1963			
World Health Organization	WHO, (in press)	Monochlorobenzene	MCB	3 ug/L*	rat-NOAEL=15mg/kg/day dog-NOAEL=27 mg/kg/day	U.S. EPA, 1980(a)	1000-10000		1.0-.10 mg/day
EPA protection of human health	U.S. EPA, 1980(b)	Dichlorobenzene	DCB	400 ug/L	multiple animal species NOAEL=13.42 mg/kg/day	Hollingworth, 1956, 1958	1000	55.6	0.94 mg/day
World Health Organization	WHO, (in press)	Dichlorobenzene	DCB 1,2 1,4	0.3 ug/L* 0.1 ug/L*	rat-NOAEL=18.6 mg/kg/day	U.S. EPA, 1980(a)	1000-10000	-	0.98-.098 mg/day
Soviet Union	U.S. EPA, 1980(b)	Dichlorobenzene	1,2 and 1,4 DCB	2 ug/L*		Varshavskaya, 1968 Stofen, 1973	-	-	-
Soviet Union	U.S. EPA, 1980(a)	Trichlorobenzenes		30 ug/L	rat-NOAEL =60 ug/L rabbit-NOAEL=80 ug/L	Gurfein and Farlova, 1962	-	-	-
Soviet Union	U.S. EPA, 1980(a)	Tetrachlorobenzenes		20 ug/L		U.S. EPA, 1977	-	-	-
EPA protection of human health	U.S. EPA, 1980(a)	Tetrachlorobenzenes	1,2,4,5-TECB	38 ug/L	beagles-NOAEL=5 mg/kg/day	Braun, 1978	1000	1125	0.35 mg/L
EPA protection of human health	U.S. EPA, 1980(a)	Pentachlorobenzene	QCB	74 ug/L	embryos in utero LOAEL=50 mg/L	Khera and Villeneuve, 1975	3000	2125	1.17 mg/L
EPA protection of human health	U.S. EPA, 1980(a)	Hexachlorobenzene	HCB	0.072-7.2 ug/L (risk levels = 10^{-7}-10^{-5})	mice, hampsters	Cabral et al., 1976 Cabral et al., 1977	- -	8690	- -
Word Health Organization	WHO, (in press)	Hexachlorobenzene	HCB	0.01 ug/L (risk level = 10^{-5})	mice, hampsters	Cabral et al., 1978 Cabral et al., 1977	- -	-	- -

Notes: Exposure assumption of 2 litres of drinking water and consumption of 6.5 grams of fish and shellfish per day used in U.S. EPA criteria development.

NOAEL - no observed adverse effect level
LOAEL - low observed adverse effect level

$$\text{ADI} = \frac{70 \text{ kg (ave. wt. of man)} \times \text{NOAEL}}{\text{safety factor}}$$

* determined by olfactory and gustatory threshold levels

section 7

REFERENCE LIST

7.0 REFERENCE LIST

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